



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

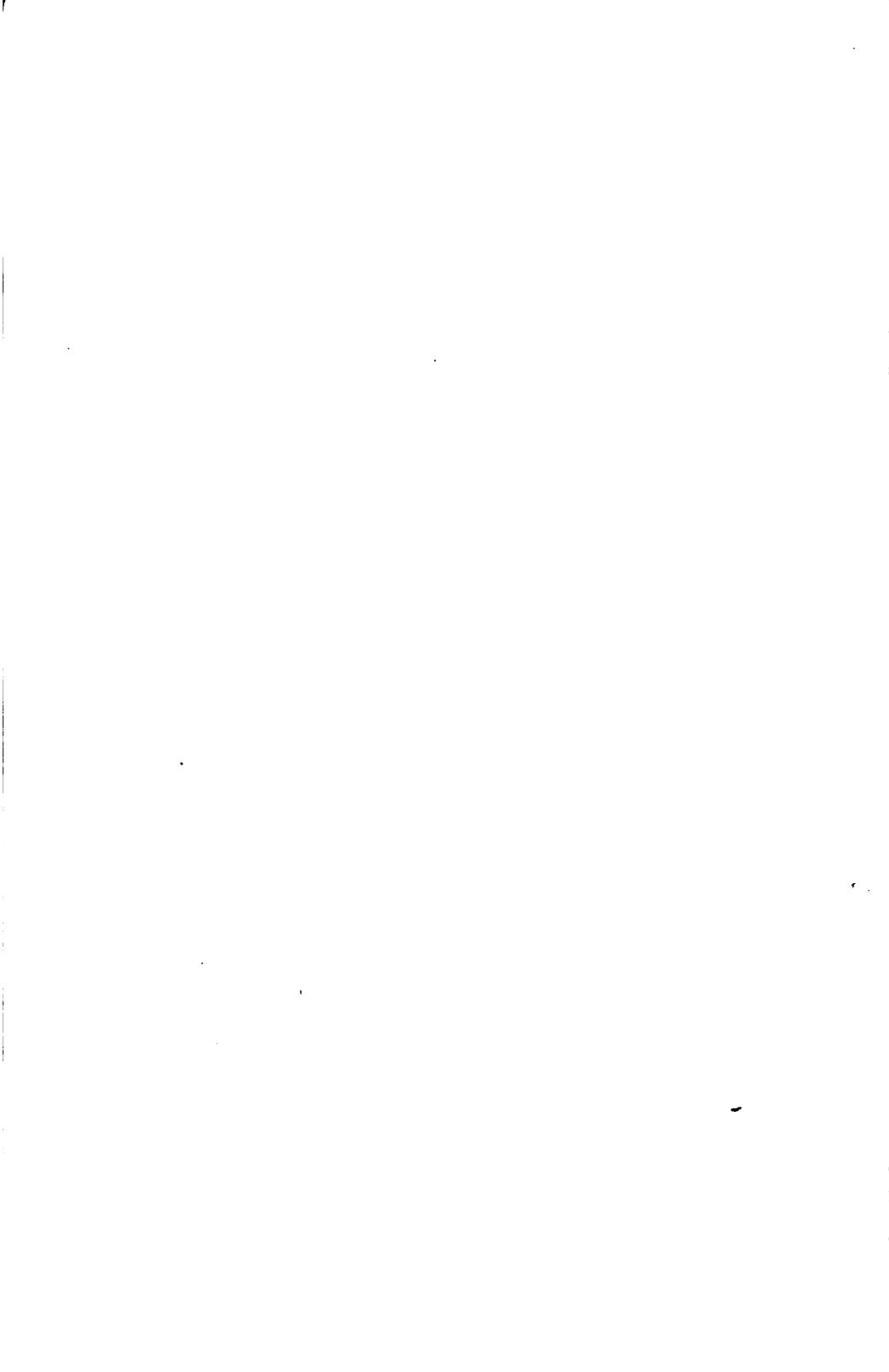
COUNTWAY LIBRARY



HC 2V19 T

6.A.55.





ESSENTIALS *of* MEDICINE

A

TEXT-BOOK OF MEDICINE

FOR STUDENTS BEGINNING A MEDICAL COURSE,
FOR NURSES, AND FOR ALL OTHERS INTER-
ESTED IN THE CARE OF THE SICK

BY



CHARLES PHILLIPS EMERSON, M.D.

LATE RESIDENT PHYSICIAN, THE JOHNS HOPKINS HOSPITAL; AND ASSO-
CIATE IN MEDICINE, THE JOHNS HOPKINS UNIVERSITY

ILLUSTRATED BY THE AUTHOR

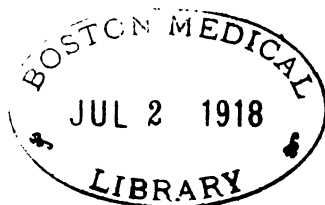


PHILADELPHIA & LONDON

J. B. LIPPINCOTT COMPANY

COPYRIGHT, 1908
BY J. B. LIPPINCOTT COMPANY

15348



*Electrotyped and printed by J. B. Lippincott Company
The Washington Square Press, Philadelphia, U. S. A.*

TO
DR. HENRY MILLS HURD
WHOSE FRIENDSHIP
IS ONE OF THE GREATEST PRIVILEGES
OF THOSE IN THE SERVICE
OF THE JOHNS HOPKINS HOSPITAL

PREFACE

SEVERAL years' experience as a teacher of medicine in a medical school and medical lecturer to nurses has convinced the writer that there is a great need for a book similar to the one which he now respectfully submits to the reader.

Many American medical students lack perspective in their medical studies. They do not learn the A, B, C of the disease first and then proceed to its more difficult study.

During their second year they are taught the pathology of a disease, including a discussion of the nature of the disease as a whole and its most difficult points. During the next year they hear much of its clinical chemistry and microscopy, and more of the theories concerning it. In the fourth year perhaps they see their first patient with that disease. They read up at once, often in a large system of medicine, all about its symptoms, course, clinical varieties, complications, sequelæ, and treatment. And so it is no wonder that if at the end of the fourth year, in a quiz, the instructor asks a very simple question about that disease, they look confused. Ask about some difficult and worthless theory and they can talk at length. If he demands a definition of that disease in twenty words for instance, they look dazed. Ask for its four most important symptoms and the four they give will often be the disputed, the accidental, or the rare ones. They have not learned to separate the important from the unimportant. Our nurses often know a great deal in a general, indefinite, inaccurate way. They seldom have a clear sharp mental picture of the elements of a subject.

It is for these two groups of readers especially that this book was written, in the hope that from it those beginning the study of Medicine and nurses may gain as their first impression a clear if limited idea of a subject.

This book may be attractive to the general reader. It will aid him to understand more clearly the medical problems of the day, to appreciate more highly a well trained practitioner, and to coöperate better with his doctor. In this manner it will fulfill the purpose of its author, and likewise will serve the need of the lay-reader far better than if he attempts to use it as a family physician, replete with information concerning the diagnosis and treatment of his ailments, an office it is not intended to fill.

CHARLES P. EMERSON,
Supt., Clifton Springs Sanitarium,
Clifton Springs, N. Y.

CONTENTS

	PAGE
INTRODUCTION.....	7
CHAPTER I.	
DISEASES OF THE BLOOD AND BLOOD-VESSELS.....	11
CHAPTER II.	
DISEASES OF THE HEART.....	49
CHAPTER III.	
DISEASES OF THE RESPIRATORY ORGANS.....	65
CHAPTER IV.	
DISEASES OF THE UPPER ALIMENTARY TRACT.....	85
CHAPTER V.	
DISEASES OF THE INTESTINE.....	108
CHAPTER VI.	
DISEASES OF THE LIVER, GALL-DUCTS, AND GALL-BLADDER.....	129
CHAPTER VII.	
DISEASES OF THE PANCREAS.....	144
CHAPTER VIII.	
DISEASES OF THE KIDNEYS.....	148
CHAPTER IX.	
DISEASES OF THE NERVOUS SYSTEM.....	183
CHAPTER X.	
DISEASES OF GLANDS.....	216
CHAPTER XI.	
CONSTITUTIONAL DISEASES.....	220
CHAPTER XII.	
THE SPECIFIC INFECTIOUS DISEASES.....	229

	PAGE
CHAPTER XIII.	
OTHER DISEASES CAUSED BY BACTERIA.....	300
CHAPTER XIV.	
ACUTE DISEASES OF UNKNOWN ORIGIN.....	307
CHAPTER XV.	
ANIMAL PARASITES.....	325
CHAPTER XVI.	
THE TEMPERATURE, RESPIRATION, AND PULSE.....	341
CHAPTER XVII.	
SIGNS AND SYMPTOMS.....	352
INDEX.....	373

LIST *of* ILLUSTRATIONS

	PAGE
1 Vegetable cells from root of Crown Imperial.....	8
2. Tip of a rapidly growing plant root.....	8
3. Transition epithelium from the human body.....	8
4. Diagram of a single cell.....	9
5. A cross-section of a tiny artery.....	9
6. Two test tubes containing blood.....	12
7. Red blood-cells.....	17
8. Finely granular leucocytes.....	20
9. A, B, "The History of a Boil".....	21
9. C, D, "The History of a Boil".....	22
10. Coarsely granular and nongranular leucocytes, and myelocytes...	24
11. Blood-platelets.....	26
12. Diagram of the circulation of the blood.....	35
13. Diagram of the two hearts.....	36
14. Diagram of an artery, capillary and vein.....	39
15. A capillary and the cells it serves.....	40
16. A small artery plugged by a thrombus.....	41
17. Two anastomosing arteries.....	41
18. End-arteries and an infarct.....	41
19. Anastomosing arteries.....	42
20. An embolus plugging a small artery.....	42
21. Arteriosclerosis of a small artery.....	44
22. Arteriosclerosis.....	45
23. Saccular aneurisms.....	46
24. Fusiform aneurisms.....	47
25. The vegetations of acute endocarditis.....	49
26. A graphic representation of the normal heart sounds.....	54
27. A diagram of the pulse waves.....	54
28. A diagram of a normal and a dilated ventricle.....	55
29. A diagram of cardiac force.....	56
30. The aortic valve.....	57
31. The pulse waves of a case of aortic stenosis.....	57
32. A pulse tracing of a case of aortic insufficiency.....	58
33. A graphic representation of the heart sounds in a case of aortic insufficiency.....	59
34. The mitral valve.....	59
35. Pulse tracing of a case of mitral disease.....	60
36. A graphic representation of the heart sounds in a case of mitral insufficiency.....	60

	PAGE
37. Internal respiration.....	65
38. The simple lungs of amphibians.....	66
39. One of the tiny lungs of which our lungs are composed.....	67
40. The bronchial tree.....	68
41. External respiration.....	69
42. Ciliated epithelium.....	69
43. A vertical section through the nose.....	70
44. Cross-section through the nostril.....	71
45. Vertical section through the skull.....	74
46. Vertical section through the skull showing a mass of adenoids...	75
47. The throat.....	77
48. A diagram of the normal thoracic and upper abdominal organs..	79
49. Emphysema of the lung.....	82
50. Lungs the seat of bronchiectasis.....	83
51. A diagram showing the position of various organs.....	86
52. The normal stomach, duodenum and gall ducts.....	89
53. Dilatation of the stomach.....	95
54. Ulcers of the stomach.....	98
55. Cancer of the stomach at the pylorus.....	101
56. A section through the wall of the jejunum showing the folds of the mucosa and the villi.....	112
57. Hernia, or "rupture".....	118
58. Diagram of a peristaltic wave.....	119
59. Intussusception.....	120
60. Intestinal obstruction caused by a twist of a loop.....	121
61. The normal cæcum showing the ileo-cæcal valve and the appendix vermiformis.....	123
62. Chronic obliterative appendicitis.....	124
63. Acute appendicitis.....	125
64. Acute appendicitis and spreading peritonitis.....	126
65. A small fragment of the liver.....	129
66. One liver lobule, vertical section.....	130
67. One liver lobule, cross-section.....	131
68. Outline of the stomach, duodenum and bile ducts, showing the various possible positions of gall-stones.....	134
69. The outline of a normal and of a "hob-nail" liver.....	136
70. Diagram of the stomach showing the venous circulation at the lesser curvature.....	137
71. Diagram of the stomach showing the venous circulation of the lesser curvature in a severe case of cirrhosis of the liver....	138
72. Diagram of an abscess of the liver perforating through the lung into a bronchus.....	142
73. Diagram of the pancreas and neighboring organs.....	144
74. Casts found in the urine.....	154
75. Upper end of a convoluted tubule together with a glomerulus ...	155

LIST OF ILLUSTRATIONS

xi

	PAGE
76. The "unit" of the kidney	156
77. Scheme of one pyramid	157
78. A "simple" kidney with only one pyramid	158
79. Diagram of a human kidney	159
80. The abdominal organs, rear view	160
81. Diagram of the urinary organs	161
82. Urinometer	162
83. Chronic interstitial nephritis	174
84. Hydronephrosis	176
85a. A cross-section through a tip of the brain	184
85b. A cross-section through the spinal cord	184
86. A cross-section of the left hemisphere of the brain through the motor area, showing the various positions of brain hemorrhages	185
87. Infarct of the cerebrum, or "softening of the brain"	186
88. Side view of the brain, the motor tract	187
89. Cross-section of the spinal cord showing the sensory tracts	190
90. A cross-section of the spinal cord showing the motor tracts	191
91. Scheme of the motor path	195
92. Cross-section of the normal spinal cord showing its anatomy ..	196
93. Diagram of the motor tract	197
94. Cross-section of the spinal cord of a case of anterior poliomyelitis	198
95. Cross-section of the spinal cord of a case of combined sclerosis ..	201
96. Cross-section of the spinal cord of a case of syringomyelia	202
97. Cross-section of the spinal cord of a case of locomotor ataxia ..	203
98. The organs of the throat	216
99. Vegetable parasites; bacteria; cocci	232
100. Vegetable parasites; bacteria; bacilli	233
101. Pathogenic organisms	235
102. The characteristic lesion of typhoid fever	238
103. The lung in acute lobar pneumonia	249
104. Sketch of the lungs of a case of double lobar pneumonia	250
105. The lungs in tuberculosis	257
106. Diagram of the chest showing tuberculosis of both lungs	258
107. Diagram of the chest showing pleurisy with effusion	276
108. Diagram of the chest showing pneumothorax	277
109. Diagram of the chest of a case of pericarditis with effusion	278
110. A cross-section of the spinal cord from a case of epidemic cerebro- spinal meningitis	283
111. Some of the smaller animal parasites	325
112. The malaria parasite	328
113. Some of the larger animal parasites	336
114. Some other important vegetable parasites	339
115. Scheme of a telephone system and of our sense organs	357
116. Scheme of our sense organs	359
117. A diagram of our sensations	363

INTRODUCTION

WHEN we aid our eyes by the microscope, a new world opens before us. Myriads of tiny creatures, living and active, swarm where they had not been suspected, while the larger animals and plants present an aspect unlike that which meets our naked eye. We find that living tissue is not homogeneous in structure, but composed of myriads of tiny units, "cells," each cell really an individual, which cohere in sheets or in masses. Let us suppose that we had never seen a brick building, and were now to get our first glimpse of one from a distance. Its walls would seem composed of a uniform red substance. As we came nearer, we should see that it was made up of many individual bricks, all similar in size and appearance, and firmly held together by mortar. At first glance "near to," vegetable tissue looks somewhat (Figs. 1, 2, 3) like a brick wall or a tiled floor, except that in the case of the vegetable tissue the "bricks" are hollow, with conspicuous walls surrounding a cavity filled with a semifluid substance. For this reason the tissue resembles a honey-comb more than a brick wall. The "bricks" of which animal and vegetable tissue is made are the living "cells," first described as the "units of structure" by Schwann in 1839. Each cell (Fig. 4) is a cavity enclosed by a perfect "cell-wall," and filled with a semifluid matter, the "protoplasm," in which is a solid body, the "nucleus." Since the first conception was that of a small, closed cavity, the most suitable name was "cell." Later it was seen that not the cell-wall but the semifluid contents, "the living protoplasm," was really the important part of the cell. In animal tissue the cell-wall is often very thin or not even discoverable, yet the name "cell" is applied here also.

The animal body consists of organs,—that is, of structures designed to perform one or more special functions. The brain, liver, muscles, etc., are organs. Each organ consists of vast numbers of cells.

INTRODUCTION

Not all cells have the same function, nor do all look alike; but those which have the same function do look alike, and are grouped together in masses which are called "tissues." For illustration, a muscle is an organ whose function is to contract. The real muscle fibres are the "muscle tissue." These are bound into bundles by fibres of "connective tissue."

FIG. 1.

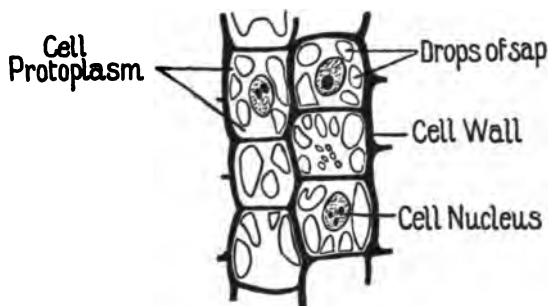


FIG. 2.

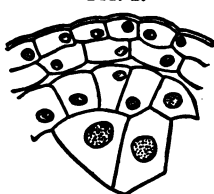
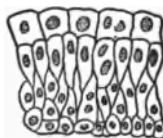


FIG. 3.



FIGS. 1, 2 and 3. Three tissues showing the "brick-wall" arrangement of the cells. (Much magnified.) FIG. 1. Vegetable cells with thick walls from the cortical layer of the root of the Crown Imperial. (Modified from Hertwig.) One can easily see why the word "cell" was first used. FIG. 2. The very tip of a rapidly growing plant root. FIG. 3. Transitional epithelium from the animal body. A cross-section of the membrane lining the bladder. The upper row of cells is part of the exposed surface.

"Elastic tissue" furnishes strength and elasticity to an organ. "Nervous tissue" is the important part, but constitutes by no means the whole, of the nervous organs.

Tissues, or masses of similar cells, are the building materials of the body. For instance, a house is built of several materials: brickwork, which is used in the walls, the chimney, the fireplaces, etc.; iron, which occurs as framework, railing for the balcony, etc.; wood, which is found in floors, window-frames, and doors. Yet, brickwork, wherever it is found, is always composed of single bricks. However much the wood-work as a whole may differ, it is all composed of many single

sticks of wood. The single bricks are the units, the wall of bricks is the tissue. So in the body there are many tissues, and these tissues are composed of single cells. Some tissues occur in but one organ, as the liver tissue, pancreas tissue, kidney tissue, etc. Some tissues occur in several organs, as connective tissue, elastic tissue, nervous tissue, etc. Figure 5 represents an artery cut across.

An artery is an organ formed to conduct blood flowing under pressure. We see here

several tissues, each composed of many cells. Inside, the artery is lined by the "intima," A, a single row of endothelial cells. This is a suitable surface for the blood to flow against. Then comes the "media," B, a thick muscle wall upon which the strength of the artery depends, and which

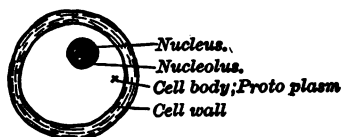


FIG. 4. A diagram of a single cell.

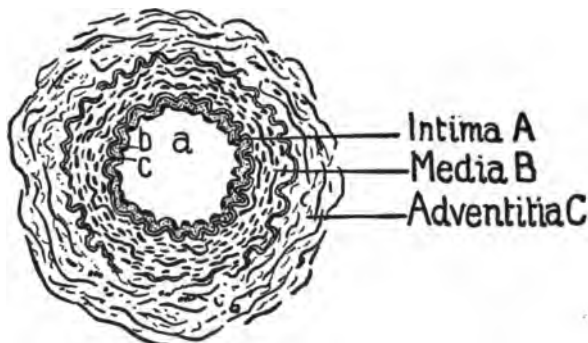


FIG. 5. A cross-section of a tiny artery. (Much magnified.) The three concentric layers of the wall are, the intima A, the media B, and the adventitia C. The lumen, in which the blood flows, is indicated by a, while b is the layer of flat cells which lines the lumen, and c the strong membrane of elastic tissue between intima and media.

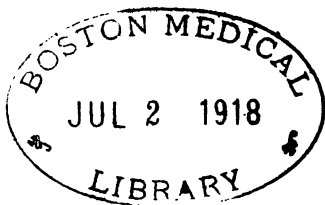
determines the calibre of the vessel. When these muscle cells lengthen, the artery's "lumen,"—its internal circumference,—increases; when they shorten, that is, contract, the artery is narrowed. Among these muscle cells is a meshwork of elastic-tissue fibres which add much strength to this muscle wall. The outermost layer is the "adventitia," consisting of bands of connective tissue, which bind the artery to the structures through which it passes.

All the cells of a tissue—all the liver cells, for example—are practically the same in size and appearance; those of different tissues are somewhat or very different. Liver cells, for instance, are quite unlike pancreatic cells or brain cells.

A cell is, in a sense, an independent structure, of which its nucleus is, so to speak, the "brain" of the cell—that on which the cell's life and activity depend. The protoplasm is the cell's "body."

Each cell has one or more functions, usually of a very special nature, to perform, and nearly all cells are capable of considerable activity. Some work almost continuously, as the kidney cells; some intermittently, as the muscle cells and cells of the stomach wall; and some serve as scaffolding, binding parts of an organ together, as the connective-tissue cells. The work of one kind of cells differs from the work of another kind. The work of the muscle cells is to change shape; the renal cells separate impurities from the blood; the gland cells manufacture new substances from the raw materials from the blood. (Some of the gastric cells, for instance, make pepsin.) Each cell while in activity is a small furnace which burns fuel, and hence produces heat. Each cell is constantly wearing out, and needs new material with which to renew its structure. Each cell lives and dies; each demands its quota of food and oxygen, and will starve or suffocate if deprived of these; each must get rid of its excreta, or "ashes," and will die if these accumulate. If one cell dies, another in its neighborhood may grow, then divide into two cells, and thus replace it. Some cells can move, most cannot. Each cell has its own individual work to do, and its success in doing it depends on its health.

Thus each organ is built of tissues, and each tissue is a colony of tiny individuals. Our body is a confederation of these colonies. Each organ has fairly separate, definite functions, the sum of the activity of all its cells, to perform, and it performs these for the whole body. This division of labor may be likened to that in the City of Baltimore. We have here carpenters, masons, tailors, shoemakers, etc., each man in a trade-union, each with a special work, which alone he can do well, each depending on others for those supplies which, by the division of labor, it is the special work of others to furnish him, and each working for the good of the whole city.



ESSENTIALS *of* MEDICINE

CHAPTER I

DISEASES OF THE BLOOD AND BLOOD-VESSELS

THE BLOOD

The Blood is the fluid, about five quarts in total amount, which is constantly circulating through the body in a system of closed tubes, or "blood-vessels,"—the "arteries," "veins," and "capillaries." Allow a little blood to stand in a vessel, and soon it is no longer a fluid but a rather solid, jelly-like mass. That is, the blood has "clotted;" "coagulation" has occurred. Soon this red "clot" begins to contract, and in so doing squeezes out drops of a clear yellow fluid, the "blood-serum." The amount of serum increases as the clot shrinks to smaller and smaller dimensions, until the clot is about one-half its original volume (Fig. 6). Thanks to coagulation, any blood-vessel that is cut or ruptured is soon closed up by the clot in the wound, and the loss of blood, or "hemorrhage," is checked. In some persons the blood will not clot, and even a small cut may lead to a fatal hemorrhage. Normal blood clots whenever it touches a foreign body or is exposed to the air. Before coagulation the blood consists of the "plasma," a limpid, straw-colored fluid, in which swarm the "blood-cells" or "blood-corpuscles;" after coagulation it consists of the serum and the clot. The latter consists of the blood "corpuscles" entangled in the meshes of a network of "fibrin," a stringy substance formed in the blood-plasma. *Blood-plasma*, therefore, is the fluid part of the blood before it clots, and *blood-serum* the fluid part after it clots.

By *blood-corpuscles* we mean blood-cells. They are of three varieties,—the red blood-cells, which give the red color to the blood, the white cells, or leucocytes, and the platelets.

THE FUNCTIONS OF THE BLOOD may be stated briefly as follows: to provide food and oxygen to the cells of all parts of the body; to remove the waste material and the carbon dioxide from these same cells; to equalize the heat of the body; and to distribute the tissue lymph, the "atmosphere" in which all tissue cells lie. In addition, it contains the internal secretions upon which much of the health of the body depends,

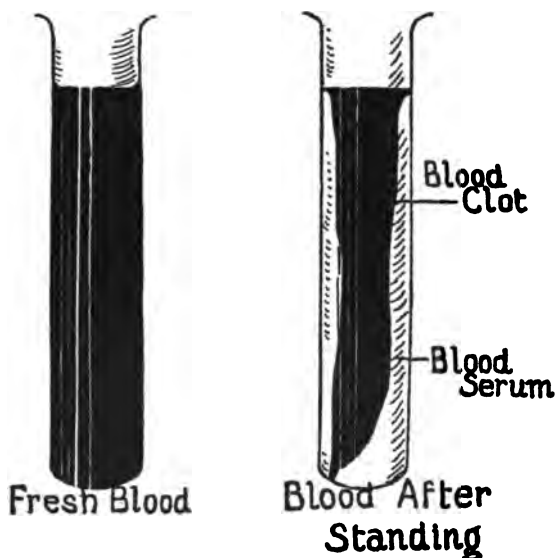


FIG. 6. Two test-tubes containing blood. The one to the left contains fresh blood. The blood in the one to the right has clotted.

and various protective bodies which prevent "blood-poisoning." The blood ministers to the health, comfort, and protection of the entire body.

To provide food. As has been stated, each living cell requires its quota of food and oxygen, just as does a living person. Deprive it of these, and it will die by starvation or suffocation. It gains its food from the blood-plasma, and the oxygen from the red blood-cells. Each cell requires food of three varieties,—protoplasm, raw materials, and fuel. Because of its activity it is constantly wearing out, and hence needs new material with which to repair itself. It can repair its protoplasm only with protoplasm, and so must have

fresh protoplasm provided it, for the animal cell is practically unable to build up this complicated substance which the plant cell builds so easily. But the cell requires also raw materials with which to do its work. Some cells are designed to produce heat, and hence require fuel to burn; it is the duty of other cells to manufacture for the body special substances called secretions (*e.g.*, the stomach cells produce gastric juice, the liver cells bile, etc.), and they must have raw materials from which to make these secretions.

The cell, therefore, somewhat resembles a steam-engine. An engine, constantly wearing out, requires steel for its repair, and it requires fuel also. Or, to change the figure, the cell resembles a mill which uses raw cotton and turns out cloth. The steel becomes a part of the engine, the coal does not; neither does the raw cotton become a part of the mill. Coal and cotton are merely used up by the engine and by the mill. So from the blood the cells gain new material for their repair, fuel to burn in order to liberate energy, and raw material from which to manufacture their secretions.

To remove waste. Each cell is a tiny furnace which by burning fuel produces heat. Just as the ashes of a large engine must be frequently removed, so the little cell must have its waste carried off, and this the blood stream does. Later the kidney cells remove this waste from the blood.

To distribute the internal secretions. By "secretion" is meant the special substance which a cell manufactures. For instance, the secretion of the stomach is the gastric juice, a fluid resulting from the combined efforts of many cells. Some of them make the hydrochloric acid, some make the pepsin, some the other constituents of the gastric juice. These various substances—pepsin, acid, etc.—are not in the blood, but are manufactured by these cells from raw materials which they get from the blood. They are the secretions of those cells in the stomach wall. Gastric juice is an *external secretion* because it is poured into an open cavity of the body, in this case the stomach. Other secretions are poured into the blood stream itself, and these are known as *internal secretions*. The latter are very difficult to isolate, but their presence is easy to prove, since deprived of them the body very quickly suffers. For instance, diabetes mellitus is a disease the essence of which is that the cells of the body cannot easily burn sugar,

their chief fuel. This unused sugar accumulates in the blood and must be got rid of through the urine. But the cells cannot use sugar unless the internal secretions of the pancreas are present. It makes no difference where the pancreas is, provided its internal secretion can reach the blood. If it is successfully transplanted under the skin of the leg, for instance, the animal will not become diabetic. The external and internal secretions of an organ are often totally independent. The pancreas produces also an external secretion, which is poured into the intestine and is very important in the digestion of food. Another illustration of an internal secretion is that of the thyroid gland in the throat. Remove the thyroid, and the patient becomes dull, then stupid, and finally imbecile. His skin gets thicker and thicker, for beneath it is deposited a considerable amount of mucilaginous fluid, whence the name of the disease, "myxedema." But let this patient, even after his mind seems entirely lost, eat every day a little of the gland of some animal,—of sheep, for instance,—the internal secretion contained in this food will supply the deficit in the patient's body, and in due time a perfect recovery may take place, which will continue as long as he eats these glands.

To distribute the tissue lymph. The distribution of the water of the body is a very important function of the blood, since each cell rests in an "atmosphere" of fluid. When this distribution is disturbed, as in Bright's disease, we may have "dropsy."

To distribute heat. The distribution of heat is one of the very important functions of the blood. Most cells are little furnaces, burning fuel and hence producing heat, and the constantly high temperature of our bodies is the result. Those parts of the body where most heat is produced are the muscles, the liver, and other large glands. Were it not for the circulation, these organs would become very hot while at work, and cool when at rest. The blood becomes heated in these organs, and then flows on into other and cooler organs—both those which produce little heat themselves, as the brain, and those which lose much heat, as the external skin. In this way the entire body is kept at a fairly constant and uniform temperature.

To circulate the protective bodies. The circulation ministers to the health and comfort of all these very delicate and sensi-

tive cells, each able to do but one or a few things, and dependent on other cells to supply through the medium of the blood all of its needs. The blood would, therefore, be an ideal home for the many germs—bacteria and other parasites—which so frequently invade it. They are accustomed to live in an adverse environment, and to win in the severe struggle for existence. To these hardy individuals an environment such as our helpless body cells enjoy would be a paradise. It is necessary, therefore, for our bodies to have protective substances which will kill these germs and yet not injure the body cells. Thus it is that the germs of “blood-poisoning” are usually quickly killed in the blood stream, while other germs, more poisonous or more hardy, do grow there and win the victory over our bodies. Again, a person who has had typhoid fever, smallpox, scarlet fever, or measles very seldom has these diseases a second time. This immunity is due to certain protective bodies, “immunity bodies,” some of which may be in the plasma.

The blood performs many other very important functions. We cite the above as instances of its work.

BLOOD-PLASMA.—The blood-plasma is the fluid of the blood. When the blood is exposed to the air, “strings” of fibrine “crystallize” from it, and catch in their meshes the blood-corpuscles, thus changing the fluid blood to a solid clot from which later the serum separates. The plasma is by all means the most important part of the blood. The red blood-cells carry the oxygen, and the leucocytes are a protection to the body. Both may have much more important functions which we do not yet know or suspect, but the known functions of the blood-plasma are manifold.

The intestinal wall is the chief building point of the plasma. It is here that it receives its chief constituents, the three proteins. We eat in our food a great variety of proteins,—fish, meat, fowl, vegetables, etc. The digestive fluids break up these complex substances into much simpler substances. The intestinal wall takes these simple bodies and builds them up again into the proteins of blood,—serum albumin, serum globulin, and fibrinogen. These are the raw materials from which all cells repair their worn structures.

The same thing happens to sugars. The variety of sugars and starches that we eat is great, but in the stomach and

intestines these are practically all changed to one simple sugar, "glucose," or, since it is found in grapes, "grape sugar." This sugar is a constant constituent of our blood, and is used up by the tissues as so much fuel.

The liver also is a plasma-former, or at least plasma-modifier. All the blood flowing from the intestinal wall, and hence containing all the food absorbed by the blood-vessels there, must pass in the portal vein through the liver. This organ removes some of this newly gained food from the blood. It removes the glucose, stores it up in the liver cells as glycogen, and keeps it until it is needed. The blood which circulates through our many organs should have a small but constant amount of glucose,—not too much, for that seems poisonous; not too little, for the muscles might run out of fuel when they needed it most. And so the liver removes from the blood in the portal vein the large amount of sugar which it contains after a meal rich in carbohydrates, stores it up, and then doles it out in just the right amount to keep the percentage constant. The normal amount of glucose is about two grammes in one litre of blood, or thirty grains in one quart. That is, the amount of fuel in our body immediately available at any instant weighs only a third of an ounce.

But the blood which flows from the intestinal wall to the liver has already passed through many other organs and has accumulated their ashes. These ashes of proteid combustion, many of them ammonia compounds, are very poisonous, and if allowed to accumulate in the blood will soon cause convulsions and death. The liver removes these ashes, transforms them into urea, and returns this harmless substance to the blood, to be removed by the kidneys. The liver removes from the blood also the hæmoglobin set free from the dead red blood-cells, and with it colors its external secretion, *bile*, yellow. Many other ashes are also removed by the liver.

After the blood leaves the liver it flows to the heart, and then through the lungs, where it loses its carbon dioxide and some water, and takes on oxygen. Hence the lungs are plasma-modifiers. The blood is then pumped through the rest of the body.

Thus it is that each cell selects from the blood that which it wants, and returns to it that which it no longer needs. The selective activity of the cells is wonderful. In the stom-

ach wall, for instance, the cells pick out raw materials, and make the hydrochloric acid, pepsin, etc., of gastric juice. The cells of the pancreas make the pancreatic juice, a very different fluid, from the same plasma. The cells of the kidney pick out only the ashes, and leave all the rest. The muscle cells pick out the fuel necessary for their contraction. The brain cells are very fastidious in their tastes; each cell takes out only what it needs from a plasma, the same for all, but how different the products of their activity! They are able to do this because they are alive; it is not a matter of physical and chemical laws as we now understand these laws. And how sensitive some of our cells are! If the circulation of the kidney is cut off for forty-five seconds, the kidney cells will not work for nearly an hour, and the poor urine they first excrete is proof of the amount of injury done by this temporary starvation.

RED BLOOD-CELLS. — In the plasma swim the red blood-cells (Fig. 7), small coin-shaped bodies, round on their flat surfaces, *a*, and slightly like a figure 8 when

seen on edge, *b*. They are really of a yellowish color, and it is only when seen in large masses that they look red. They are so small that 3300 of them lying on their flat surfaces in a line, *c*, with their edges touching, will reach only one inch, and it would take 16,500 piled one upon another like coin, *d*, to make a column an inch high. There are so many of them in the blood that a drop about the size of the head of a small pin will contain about 5,000,000 cells.

The duty of these cells is to carry oxygen. They do this by means of hæmoglobin, a very interesting pigment which forms 95 per cent. of their composition. Of this hæmoglobin there are about 14 grammes in every 100 c.c. of blood, or about 1½ pounds in the entire body. Hæmoglobin contains iron, and it is because of this iron that it is able to carry oxy-

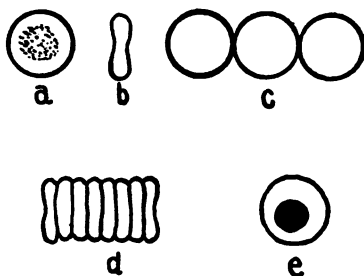


FIG. 7. Red blood-cells. (Magnified 1000 times.) *a*, shows the flat surface of one cell. The shadow in the centre is evidence of its biconcavity; *b*, one cell seen from the edge; *c*, three cells lying flat, their edges touching; *d*, several cells in a "rouleau," or piled like coin; *e*, a "normoblast" or red blood-cell with a nucleus.

gen. The iron is small in amount: there are only about three grammes, or a tenth of an ounce, in the entire body. The hæmoglobin of some of the smaller animals, as the squid, has copper instead of iron, and their blood is blue instead of red. The green pigment of plants, chlorophyll, is similar to hæmoglobin, and also contains iron.

The framework of each red blood-cell is called its "stroma." This is small in actual amount, for the most of the cell is hæmoglobin, and yet to it the cell owes its solidity, size, shape, and elasticity. If all the hæmoglobin is washed out of the cell, this stroma will preserve the exact shape and size of the corpuscle. This process of washing the hæmoglobin out of the cell is known as "laking." The blood can be laked in many ways. For instance, if the cells are poured into pure water, the hæmoglobin is at once dissolved out of them. It is to avoid this, as all nurses know, that we inject into the body, as an infusion or transfusion, not pure water but physiological salt solution (that is, water, with 6 grammes of table salt per litre). Many poisons will lake the blood, as snake poison, and the poison of many germs. Even the blood of one animal may be poisonous to another. This is the reason why the transfusion of blood from a healthy animal to a sick man was abandoned; although theoretically good, the blood may kill the man. We do transfuse, but only from man to man, and then feel anxious unless the two persons are blood-relatives. Chemically, so far as our present tests go, many bloods are the same; but physiologically, that is, judged by their effect upon the life or the health of animals, they are very, very different. The serum of a dog, for example, is so poisonous to a rabbit as to kill him.

Hæmoglobin has an attraction for oxygen, and when hæmoglobin and oxygen are brought together they unite and form oxyhæmoglobin, which is very red. But this combination is very weak, and oxyhæmoglobin readily gives up its oxygen if the surrounding air or fluids have less oxygen than itself. Hæmoglobin has a bluish-red color, whence the difference in color between arterial and venous bloods. This affinity for oxygen is the secret of respiration. In the lungs the hæmoglobin of the red blood-cells is exposed in a thin layer to the air, which contains about 20 per cent. of oxygen. Oxygen and hæmoglobin unite and form oxyhæmoglobin.

This blood then circulates back to the heart, and thence around through the tissues. The tissue-cells are meanwhile using up the oxygen they had, and all they can get from the lymph around them. Hence this lymph is poor in oxygen, and so has a stronger affinity for it than has the hæmoglobin in the blood-vessels. The oxyhæmoglobin at once splits, its oxygen goes to the tissues, and the blood then circulates back through the veins to the lungs for more oxygen. The carbon dioxide which the cells give off is carried by the plasma to the lungs. There is very little carbon dioxide in the air of the lungs and so the carbon dioxide leaves the blood and takes the place in the air of the oxygen which the hæmoglobin is absorbing. That is why the air we exhale is "bad." This interchange, within the blood, of oxygen for carbon dioxide is known as "internal respiration;" the interchange in the lungs is known as "external respiration."

The red blood-cells are formed in bone-marrow, in the case of adults chiefly in the marrow of the ribs. As any housewife knows, there are two kinds of bones,—those in which the marrow is red and nutritious, "marrow bones," and those which contain only fat, and hence are of no value as food. It is in the red marrow that these red blood-cells are formed. They have no definite cell membrane, but when young have, like all perfect cells, a nucleus (Fig. 7, *e*). This nucleus, however, is not necessary in respiration, and would probably make the cell too heavy. It is, therefore, by a wise provision, discarded as soon as the cell is old enough to be used in the circulation. The red blood-cells of reptiles, birds, and some animals—camels, for instance—all have nuclei. The red blood-cells which have lost their nuclei have lost, therefore, their most important element, so far as cell life goes. They have sacrificed their life for the sake of their function. Sometimes when the blood is very much in need of new cells, as after a severe hemorrhage, these young nucleated reds are allowed to enter the circulation; but this is not normal, for naturally they remain in the bone-marrow until old enough to lose their nuclei. Red cells remain in the circulation perhaps about three weeks. Interestingly enough, we can find in normal blood no old, or half-worn-out cells, for before the first sign of age appears the cells are removed and destroyed, perhaps by the spleen, the bone-marrow, and the

liver. The pigment which gives the color to bile is made from the hæmoglobin of these worn-out cells. After the liver has carefully split off the iron, and saved it for further use, the rest of the pigment is "thrown away" in the bile.

We see, therefore, that the one function of these red blood-cells is to carry oxygen, a very necessary but still rather simple function, while the functions of the plasma are much more numerous and complicated. And yet, in studying the

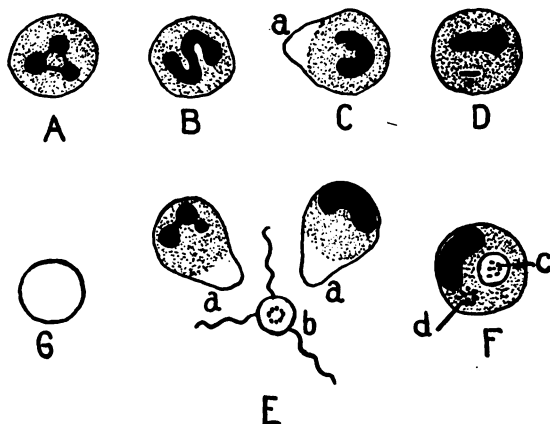


FIG. 8. Finely granular leucocytes. (Magnified 1000 times.) *A, B*, two leucocytes at rest. *C*, a leucocyte moving. Note its "pseudopod," *a*, of clear protoplasm and that the inert granules and nucleus "collect in the rear." *D*, a leucocyte which has "swallowed" a bacillus which it is now digesting. The clear halo around the bacillus represents the digestive fluid which the protoplasm has secreted. *E*, represents two leucocytes both racing for the same flagellated malarial parasite, *b*; *a* and *a* are the pseudopods of the leucocytes. *F*, a leucocyte which has swallowed two malarial parasites, *c* and *d*, and is now digesting them. The malarial parasite *c*, is possibly still alive, but only a few pigment granules are left of *d*. *G* is a red blood-cell introduced in the picture for comparison of size. Note the variety of shapes of the nuclei of the seven leucocytes.

blood in anæmia, we judge its condition chiefly by the number and condition of the red blood-cells, since they are much easier to study than is the plasma, and are, evidently, a very sensitive index of blood conditions.

LEUCOCYTES.—The leucocytes are also blood-cells, but contain no hæmoglobin, and hence are colorless. They are "perfect" cells. They have a nucleus; they are alive, and able to live a rather independent existence, they move about of their own volition and rapidly, too. Normally there is about one leucocyte to 500 of the red cells; that is, there are about 7000 of them in each cubic millimetre of blood. They

are not flat cells, like the reds, but are usually spherical, or fairly so, and they can change their shape rapidly.

The majority of leucocytes—that is, from 70 to 72 per cent. of them—are about one-third larger than red blood-cells, (Fig. 8). Their nucleus, may be of almost any shape except round, and their protoplasm is filled with very fine, dust-like granules. These cells move by projecting their protoplasm at one point, *a*, and then flowing into this pro-

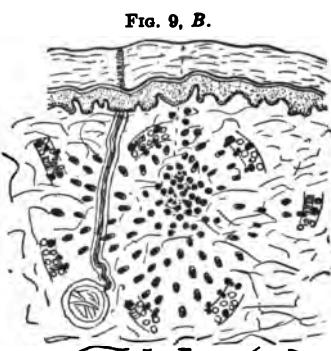
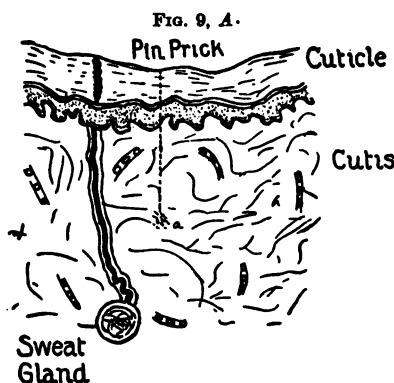


FIG. 9, A. "The History of a Boil." This figure represents a cut-section of normal skin. Note the surface layer, or cuticle, and the "true skin," or cutis. In the cutis one sees that the blood capillaries are just wide enough for the blood-cells to pass through "in single file." The skin has just been pricked by a dirty pin. On the point of this pin were several poisonous germs which were deposited at *a*.

FIG. 9, B. "The History of a Boil" (continued). The poison from these germs (*A, a*) diffuses through the cutis. The capillaries dilate. The leucocytes force their way through the walls of the capillaries and travel towards these germs. Note the dumb-bell shape of the leucocytes as they pass through the minute holes in the capillary walls, and their pseudopods as they travel towards their common destination, attracted by the poison from the germs. The skin in this region is now swollen, red, hot, and painful.

jection, which is called a "pseudopod." They live on that which they absorb from the surrounding plasma, but when they come across any solid body that attracts them, a germ for instance, they "swallow" it by enclosing it in their substance (Fig. 8, D). The protoplasm touching the germ secretes a digesting fluid, which forms a bubble around the germ, and one can watch the latter fall in pieces and disappear as it is digested. This process of devouring a smaller cell or germ is called "phagocytosis." The leucocytes seem able to form digesting fluids which differ according to the tasks they have to perform. These cells are the scavengers of the body. If any dust or dirt, (Fig. 8, *d*), get into the blood, it is their

duty to pick it up and carry it to the ash-heaps of the body, —spleen or bone-marrow. The malaria germ, for instance, digests the hæmoglobin of the red cells in which it lives, and in so doing forms masses of fine dust-like refuse, which later become free. The leucocytes pick these up and carry them to the spleen, where they can be found years later. Again, the leucocytes are the policemen of the body. Should a germ get into the blood they will try to pick it up and digest

FIG. 9, C.



FIG. 9, D.



FIG. 9, C. "The History of a Boil" (continued). The migration of leucocytes has continued until now they form a dense mass surrounding the germs. The poison of the germs has killed all the leucocytes and also all the cutis immediately around them, and now digestive fluids from the dead leucocytes is turning the whole dead mass into liquid pus. The boil has "come to a head." There is a little lump on the skin and through its thin covering of cuticle can be seen the yellow pus.

FIG. 9, D. "The History of a Boil" (concluded). The boil has finally ruptured. The liquid pus has escaped carrying with it the germs and most of their poisons; the migration of leucocytes has stopped; the capillaries are returning to normal size and now new tissue will grow and fill up this hole.

it (Fig. 8, D, E, and F). If the germ produces a very strong poison, many of them will probably die in the attempt. Leucocytes will wander out of the capillaries into the tissues, and there they are called "pus-cells." Supposing one pricks his finger with a pin which has virulent germs on it (Fig. 9). A. These germs, *a*, are deposited under the skin, where they form poisons which kill many of the cells in the neighborhood. These poisons become diffused through the tissue. B. The capillaries in the neighborhood at once become distended, and the leucocytes of the blood collect in them, wander through their walls, and move from all sides towards the infected area. C. They form a solid wall, around these germs, and

seem to try to kill them by devouring them or by setting free "germicidal substances" in their neighborhood. At the same time the dead bodies of the pus-cells give off a digesting ferment which liquefies the dead tissue. The finger at first was uniformly swollen, hot, red, and painful; now this collection of leucocytes makes a projection at one point. The surface at this one point soon becomes yellow, since the skin there is dead, and since below it is a large mass of these pus-cells. When the digestive process has reached the skin we speak of the boil as "coming to a head." We mean that the ferment has digested the tissue from the spot where the bacteria were through to the skin. *D.* This soon ruptures, and the pus oozes out. The pus consists of all this liquefied tissue, the bacteria, their poisons, and the great masses of pus-cells. Then if all the germs are gone the hole is soon filled in by new tissue, and the boil is healed.

The process of engulfing a germ is called "phagocytosis." It is best studied in malaria, (Fig. 8, *E*). One finds under the microscope an active malarial germ. An active leucocyte in the neighborhood will very soon move towards it, swallow it into its substance, and digest it. Sometimes two will come from opposite directions; three or even four leucocytes may be seen racing, as it were, to see which can capture the enemy. This attraction is undoubtedly due to a poison from the germ which is diffused in all directions, and which, when it reaches the leucocyte, attracts it. This attraction is known as "positive chemotaxis." "Negative chemotaxis" occurs when the poison is so strong or of such a nature that the leucocyte moves as fast as it can in the opposite direction, as if to escape.

Leucocytes not only fight for the protection of the body, but also carry the fat in tiny globules around the body.

The leucocytes described above are filled with very fine granules, and hence are known as "finely granular cells." Since their nuclei are never spherical, but of various other shapes—those of strings fantastically twisted, rods, balls, etc.—they are also called "polymorphonuclears," whence their whole name, "polymorphonuclear-finely-granulars," or, since their granules are stained by neutral dyes, "polymorphonuclear neutrophiles." There is another group of leucocytes (Fig 10, *A*), similar in size and in nuclei, but different in the

size of the granules, which are very coarse. These, therefore, are called the "polymorphonuclear-coarsely-granular cells," or, since these granules are stained by a dye named eosin, "polymorphonuclear eosinophiles." These cells are normally only from two to four per cent. of the entire number of leucocytes. They can move more rapidly than do the finely granular cells, and they seem to be an advance guard, for when trouble arises they are usually first on the field.

In normal blood from 20 to 25 per cent. of the leucocytes are the "small mononuclears" (Fig. 10, *B*). These are about

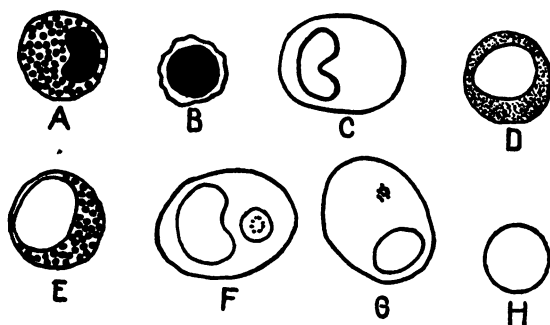


FIG. 10. Coarsely granular and nongranular leucocytes, and myelocytes. (Magnified 1000 times.) *A*, a coarsely granular leucocyte, or "eosinophile." *B*, a small mononuclear leucocyte, or "lymphocyte." *C*, a large mononuclear leucocyte. *D*, a finely granular mononuclear leucocyte, or "finely granular myelocyte." *E*, a coarsely granular mononuclear leucocyte or "coarsely granular myelocyte." *D* and *E* are never found in the blood of a normal person. They occur normally in the bone marrow. *F* and *G* are leucocytes similar to *C* which have swallowed malarial parasites. *H*, a red blood-cell.

the size of red blood-cells. They have a relatively large, usually spherical, nucleus, and a very narrow rim of protoplasm which contains no granules. They are also called "lymphocytes," since they were once supposed to come from lymph glands, while probably they do originate in the bone-marrow. They are also motile, and seem especially attracted by the toxin of the germ of tuberculosis, since in the fluid which collects in this disease these are the predominating cells.

About four per cent. of the leucocytes are the "large mononuclears" (Fig. 10, *C*, *F*, *G*), which are five or six times as large as a red blood-cell. They have considerable clear protoplasm, and large nuclei, which in shape are oval or indented.

It will be noted that no granular cells with round nuclei are found in normal blood and yet all the granular cells when young have round nuclei, but then, like nucleated reds, they stay in the bone-marrow where they are formed. When they are old enough to enter the circulating blood, their nuclei have become contracted and irregular in shape. While the nuclei are round, these cells are called "myelocytes;" and of course there are "coarsely granular myelocytes" (Fig. 10, *E*) and "finely granular myelocytes" (Fig. 10, *D*). In one disease, leukæmia, myelocytes occur in the circulating blood in considerable numbers.

Normally there are in the blood from 5000 to 10,000 leucocytes per cubic millimetre. When the count is above 10,000, the condition is called a "leucocytosis;" when below 5000, a "leukopenia." An important aid in diagnosis is not alone the total count but the relative count of these various forms, for with the same total number their percentages may be quite different. Inflammations anywhere in the body, or diseases with pus formation, cause a rise in the count, due to a great increase in the polymorphonuclear-finely-granulars. In pneumonia, for instance, the leucocytes may be even 30,000 or 100,000 or more per cmm. This increase of leucocytes means, we believe, that the person is making a good, vigorous fight against the germ of pneumonia. And yet no idea of the outcome can be gained from the count, for even with the most vigorous fight the patient may lose, that is, die. If, however, the patient is weak, and the leucocytes do not rise at all, we believe he is not strong enough to fight against it, and the outlook is very serious. We do not know exactly how the leucocytes fight an infection—whether they devour the germs or not; but we know that in pneumonia their increase is an index of the fight which the patient's tissues are making against the infection. In other diseases surely caused by germs—typhoid fever, and tuberculosis, for instance—the leucocytes, instead of increasing, decrease in total number. This difference between diseases we cannot yet explain. It is fortunate there is this difference, for it is a very great aid in diagnosis.

In other cases it is the polymorphonuclear-coarsely-granular cells that are increased. This "eosinophilia" is of great help in the diagnosis of infections with animal parasites,

especially "trichinosis." But these cells are increased by any animal parasite, even the common round worms in the intestine. They are also increased in asthma.

In malaria it is the large mononuclears that are increased, and these being phagocytic may contain pigment granules (Fig. 10, *G*), or whole malarial parasites (Fig. 10, *F*), in their protoplasm. In other conditions the small mononuclears are increased. By studying the leucocytes, therefore, we receive valuable assistance in diagnosis.

BLOOD-PLATELETS.—In the blood is found also a "third corpuscle" (Fig. 11), a colorless cell whose origin and function are much in dispute. It seems to be important in blood

coagulation, and yet even that is not certain. These "platelets," as they are called, are present to the number of about 250,000 per cmm.

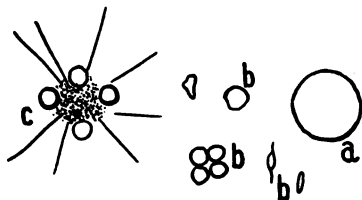


FIG. 11. Blood-platelets (magnified 1000 times). *a*, a red blood-cell introduced for purposes of comparison. *b*, blood-platelets seen from the side and edge. *c*, a mass of platelets. The most have broken down to a granular mass and from this as centre strands of fibrin radiate.

Diseases of the Blood.—

Since the composition of the blood depends on many organs, one would expect that disease of these various organs would greatly influence the blood. But it is mar-

vellous that the blood, so far as we can judge, remains remarkably normal, even when there is serious disease. For instance, a person with diabetes, instead of voiding about one quart, may void over 25 quarts of urine a day; instead of voiding with this water about $\frac{1}{4}$ of a pound of solid matter, the kidneys may void even 2 pounds of solid matter, most of which is sugar. Now, all of that water and sugar in the urine come from the blood, and yet, if the blood is carefully studied, scarcely any change in it can be detected. The most important causes of injury to the blood are the poisons produced by bacteria.

ANÆMIA.—By anæmia is meant a poor condition of the blood. Patients who are anæmic look pale,—not necessarily their cheeks, but their tongue and lips. The color of their cheeks depends upon the amount of blood flowing through the skin, not so much on its richness as does the color of these

mucous membranes. The cheeks of even an anæmic person can blush.

The volume of blood in the blood-vessels is practically always the same, for the heart is a mechanical pump, and needs a certain volume of some fluid upon which to work. If a large amount of blood is suddenly lost, as by a severe hemorrhage, the tissue lymph in which the cells lie, and which is not in the capillaries, at once flows into the vessels and makes up as far as possible for the lost volume of fluid. By anæmia we do not mean a decrease in the amount of blood in the body; we mean that its quality is poor. Some day perhaps the quality of the blood will be judged by its plasma, but at present we judge it, first, by the number of red blood-cells actually counted in each cubic millimetre of blood; secondly by the amount of hæmoglobin per hundred cubic centimetres. This is determined by diluting a measured amount of blood, in order that the color may not be too deep, in a measured amount of water, and then matching it against a standard scale. A normal man has about 5,000,000 red corpuscles in one cubic millimetre of blood and a normal woman about 4,500,000. In some anæmias the count is as low as 200,000, or even lower; in certain other conditions, as high as 10,000,000. The latter condition is known as "polycythæmia."

Hæmoglobin is expressed in terms of percentage. A normal person has about $14\frac{1}{2}$ Gm. per hundred cubic centimetres, and, therefore, this is called 100 per cent. When we say, for instance, that a person's hæmoglobin is 50 per cent., we mean that his blood contains one-half the proper amount of hæmoglobin. When the marrow must build cells with unusual rapidity, as after a hemorrhage, it produces smaller and thinner cells than the normal, that is, each cell contains too little hæmoglobin. The count may be almost normal, and the hæmoglobin very low. The relation between these two values, the percentage of hæmoglobin divided by percentage of the count, is called the "color index." For example, suppose a person's count is 4,000,000 cells, that is, four-fifths, or 80 per cent., of the normal number, and the hæmoglobin 60 per cent. Then 0.75, the result of dividing 60 by 80, is the color index. In certain other diseases the red cells are unusually large and thick. We could, therefore, have the

count 1,000,000 cells, or 20 per cent. of the normal number, and the hæmoglobin 30 per cent. The color index in this case would be 30 divided by 20, or 1.5.

SECONDARY ANÆMIA.—By secondary anæmia is meant an impoverished condition of the blood due to some cause which we know, as hemorrhage, the poison of a fever, etc. In practically all secondary anæmias the hæmoglobin is more reduced than is the count, and hence the color index is usually low. We might, for example, find a count of 4,000,000 cells and hæmoglobin of 70 per cent., with a color index, therefore, of 0.87. That is, the count may be almost anything below normal, but the hæmoglobin will usually be still lower, and the cells will look small and thin. While the blood is being restored, the over-active bone-marrow often hurries nucleated reds into the circulation; on certain days we even see vast numbers of these cells, and this "crisis," as this period is called, is followed by a jump upwards in the red count. Since the bone marrow produces leucocytes also, it may, now that it is very active, produce along with the reds a large number of leucocytes. Hence the white count is often over 10,000.

Secondary anæmias are due to hemorrhage, to certain poisons, especially those produced in acute fevers, to chronic diseases, to poor food, etc. The anæmia due to hemorrhage may be acute or chronic.

Acute Anæmia due to Hemorrhage.—By this we mean an anæmia due to one sudden loss of blood. A person can lose from one-half to two-thirds of his blood, that is, over two quarts, before his life is in jeopardy. Such hemorrhage occurs when an artery is opened by a sharp instrument, or when a very severe nose-bleed occurs, or when a small vessel ruptures, as in gastric ulcer or in a tuberculous cavity of the lungs. The blood at once makes up its volume of plasma from the tissue lymph, and then the bone-marrow very actively supplies new cells. To do this takes from one to thirty days, according to the amount of blood lost.

A chronic anæmia is one produced by a series of hemorrhages occurring at intervals so close together that there is not time entirely to restore the loss due to one before the next occurs. Such an anæmia results from repeated hemorrhages from the lungs, from ulcerated cancers, from hemorrhoids, etc. The patient may not even know he is bleeding. It takes a

much longer time than in acute anæmia, even eight or ten months after bleeding stops, to recover from such anæmia.

There are many blood poisons which injure either the blood itself or the organs which produce it. A good illustration is the "blood-poisoning" which follows a prick with a dirty pin. A few germs, *e.g.*, streptococci, get into the system, multiply in great numbers, and with their poisons rapidly destroy the red blood-cells. Another illustration is the anæmia of those who work with white lead, or arsenic. The poisons of the germs causing pneumonia, diphtheria, typhoid fever, acute articular rheumatism, produce anæmia. One of the common causes of secondary anæmia is the poison of cancer, which is very injurious to the blood; one of the commonest is tuberculosis. Certain parasites which live in the intestines, as the hook-worm, may produce an anæmia by their poisons as well as by the minute hemorrhages they cause.

The Anæmia of the Poor.—This anæmia occurs especially among the poor, but also among the rich. Some persons are anæmic because their good blood is injured by a poison, etc., but in these cases a poor blood is formed. The trouble is chiefly with the gastro-intestinal tract. Such persons perhaps do not eat good food; for instance, the poor of Ireland, who live on potatoes and black bread, which contain very little blood-building material. Good food for the blood must contain iron and much proteid, and unfortunately the meats and the green vegetables, which contain the most of these ingredients, are the most expensive. Again, the food may be good, but if it is poorly cooked it is digested with difficulty. Thus the frying pan is an important cause of anæmia. Or the alimentary canal may be unable to digest the food. Or perhaps the person does not chew his food sufficiently, but eats rapidly and works immediately after meals. His food is not in a condition to be digested, and irritates the stomach and bowel. But more important than these is the state of mind of the patient, for a person always worrying cannot digest his food properly. But, after all, the stomach and the blood will stand a great deal of abuse, and the wonder is that we are as well as we are.

Certain medicines cause very severe anæmia, especially the "headache powders" advertised as "harmless," but loaded with powerful drugs.

The above are a few causes of secondary anæmia—some rare, some more common. By far the most important and common cause for the young is tuberculosis; for the elderly, cancer.

PRIMARY ANÆMIA.—By primary anæmia we mean an anæmia whose cause is not yet known. As soon as the cause is known, that anæmia will then be classified as secondary. The primary anæmias are chlorosis, pernicious anæmia, leukæmia, and pseudoleukæmia.

Chlorosis is a disease of girls from about twelve to fourteen years old. It does occur in older women, especially in those who had it when young. It has occurred in men, but so rarely that the diagnosis is usually open to doubt. The patient may be recognized at first glance by the very pale lips, the greenish color of the skin,—hence the name “chlorosis,”—and the bluish tint of the whites of the eyes. The patients are generally well nourished, and except for their color look healthy. They complain of shortness of breath, palpitation of the heart, especially on exertion, and usually of indigestion. The blood shows an anæmia resembling the secondary type; that is, the hæmoglobin is more reduced than is the number of the red cells. The count is often almost normal, but as a rule is 4,000,000 or just above. The hæmoglobin is often between 40 per cent. and 50 per cent., and hence the color index is about 0.5. Under the microscope one sees the reason for this low index. The cells are practically all smaller than normal, and so much thinner that they look as if they had a hole in the centre, like a doughnut. The leucocytes are about normal.

The cause of this anæmia is doubtful, but it seems to be wholly due to poor blood formation and not at all to blood destruction. As it occurs at an age when there is a marked acceleration in the growth of the body, and also when the red marrow bones are many of them becoming fatty, which means a decrease in the amount of blood-building tissue, it would seem as if the tissue left were over-taxed.

The treatment is simple. These patients need to stay in the fresh air more than they do, and to eat more nourishing food; but especially do they need iron as medicine. Iron in the cheapest forms will do. The market is flooded with expensive iron preparations, organic compounds, etc., but they

have the one common characteristic of costliness, and are no better than the very cheap Blaud's pills.

Primary Pernicious Anæmia.—As the name implies, this is an anæmia the cause of which is not known, and which in almost every case sooner or later ends fatally. It occurs particularly in adults, and, since they lose very little weight, they are often robust-looking men. Their lips and tongue are rather pale, their face is of a brownish or yellowish tint which resembles sunburn, and they pass as very healthy-looking persons. That they do so is attested by the number of them that succeed in getting large insurance policies after the disease begins. These patients complain of indigestion, shortness of breath, palpitation of the heart, extreme weakness, sometimes a slight swelling of the feet, or a tingling in the feet and hands. It is interesting to observe how many different diagnoses are made in such cases. They come to the clinic with the diagnoses of heart disease, jaundice, Bright's disease, paralysis, and a puerperal neuritis, and have been treated for one or more or all of these diseases. The blood shows an extreme anæmia. The count is often as low as 1,000,000 or even 200,000, but the hæmoglobin is relatively high for such a count, perhaps 30 per cent., and hence the index is practically always over 1, even 1.75, a very important point in diagnosis. The leucocyte count is low, and there is an increase in the small mononuclears.

This anæmia is due to two causes: first, blood destruction, which is shown by the jaundice, and the very high color of the urine; and, second, the deficient production of the new cells. The body tries to protect itself by producing unusually large, thick cells. Often one sees nucleated reds, especially those larger than are any seen in the secondary anæmia. These "megaloblasts" are very important in the diagnosis.

The treatment is, above all else, rest in bed, good food in abundance, fresh air, sunlight, and arsenic in some form. We usually use Fowler's solution, which contains about 1 per cent. of the oxide of arsenic. One begins with 3 minims after each of the three meals of the day, and this dose is increased by 1 minim every third day until the dose is about 15 minims. At this stage it is well to discontinue this drug for a week, and then to begin again with a dose of 3 minims. If the patient complains of a cold in the head, or of stomach trouble, or if

the eyes are puffy, the arsenic should be stopped. If it is taken over too long a time the skin may become very dark, and in places very thick. We now very often use atoxyl, an organic preparation of arsenic, and give it hypodermically, one dose every other day—the first dose one-half grain, and each succeeding dose increased by one-fourth of one grain. The advantages of atoxyl over the oxide of arsenic are the ease with which the drug is given, and the little disturbance it produces; but whether it is more efficient or not is an open question.

The course of these cases is up and down. Some fail rapidly until death; others seem to get entirely well, and then relapse. Some of our patients have come to the clinic three or four times. Practically never is the patient cured. The cause of this anæmia is as yet unknown, but it is certainly some disease which produces a poison injuring the bone marrow.

It is very interesting to see how little difference in the symptoms the blood count makes. The symptoms do not seem to depend at all upon the number of red cells in the blood, but the low blood count itself is only one of the symptoms of the disease. The girl with chlorosis, hardly able to walk because of the shortness of breath and weakness, may have 4,000,000 cells per cubic millimetre. A man with pernicious anæmia may lead a very active life and yet have a count of but 1,000,000, or lower, and seek treatment for some minor symptom, as gastric indigestion, etc. His symptoms even when extreme may so improve that he is confident he is well although his count has scarcely risen.

MYELOGENOUS LEUKÆMIA.—By leukæmia is meant literally "white blood." The disease is so named because in extreme cases there are sometimes as many leucocytes as red cells, and the blood looks milky even to the naked eye. There is nearly always a marked anæmia,—by which we mean reduction in the number of red blood-cells,—and hence it is that the leucocytes are able to equal or outnumber the red cells, for the leucocyte count is very seldom above 1,000,000. As a rule, it is about 500,000 per cmm. Leukæmia is a disease of the bone-marrow, but affects also the spleen and the lymph glands. These become enlarged, and the majority of patients apply for treatment because they discover a "tumor" in the

abdomen, that is the big spleen. The general symptoms felt by such patients are very like those of pernicious anæmia.

In leukæmia the red blood-cells are occasionally normal in number, but usually they are diminished, sometimes much so. The leucocytes may vary from normal to even 1,500,000. But it is not the total number of white cells which is important. If it were we could not separate leukæmia from many cases of leucocytosis. The important point is that in leukæmia myelocytes are found in the blood. Those young forms of the granular cells with round nuclei, which are never present in normal blood, are in this disease found in large numbers. One finds also many nucleated red blood-cells, especially megaloblasts. Even when the total count of leucocytes is about normal the presence of these abnormal cells will give us the diagnosis.

LYMPHATIC LEUKÆMIA resembles the myelogenous form in its general symptoms, although the lymph glands are often much larger, but the blood looks very different. In the lymphatic form, as well as in the myelogenous, the total count of leucocytes is much increased. This increase, however, is not of the granular leucocytes but of the small and large mononuclear nongranular cells. One may, indeed, fail to find a single cell with granules. The white count seldom runs above 200,000 per cubic millimetre. Sometimes it is the small mononuclears only which are increased, sometimes cells like them, but larger. These are, we think, the very young forms of small mononuclears, and they also occur normally only in the bone-marrow. In this form of leukæmia the red-cell count is usually lower than in the myelogenous form. Few nucleated reds are found.

Patients with this form of leukæmia are especially liable to have hemorrhages. The course and prognosis of these two forms of leukæmia are very similar to those of pernicious anæmia, and the treatment is practically the same.

ACUTE LEUKÆMIA is a very severe, quickly fatal form of leukæmia, so rapid in its development that the disease has no time to enlarge the spleen, lymph glands, and liver. The bone-marrow alone becomes diseased. The leucocyte count is often very high, and the large mononuclear nongranular cells are the ones especially numerous. This disease is interesting because it is so often diagnosticated as diphtheria,

scurvy, purpura, typhoid fever, pernicious anæmia, etc.; and yet if one examines the blood he can often recognize the leukæmia at a glance.

HÆMOPHILIA.—This very interesting but rare disease deserves mention. It occurs in men, almost never in women. The blood of these men seems, at times at least, unable to clot, and they are called "bleeders." Wherever they are struck a large bruise forms and a small nick in the skin may cause a hemorrhage that is really serious, since the blood may ooze from this cut for days. Should a surgeon inadvertently operate on such a case he would doubtless kill his patient. These men are very careful never to shave, for fear they may cut themselves even a little. They may bleed to death from the nose, or from the mouth after a tooth has been pulled.

It is interesting to note that while hæmophilia occurs in men almost exclusively, yet they inherit it from their mothers and not from their fathers. For instance, Mr. A is a bleeder, neither his sons nor his daughters will be bleeders, but his daughters' sons will be, not his sons' sons. That is, the disease is transmitted only through the women in the family, and yet they themselves are not bleeders. For the treatment of this condition, as in all those diseases with severe hemorrhage, like purpura, etc., we give some form of calcium. Calcium is necessary in the coagulation of blood, and if we give it to a patient whose blood clots too slowly it will soon clot much faster. The best form of calcium to give is the lactate, 30 grains three times a day. Calcium lactate is scarcely soluble in water, and hence must be given as a suspension. Calcium lactate decomposes quickly, so that only fresh preparations should be used. Formerly we gave calcium chloride, fifteen grains three times a day, but this irritates the stomach, and must be given in a large volume of water.

The Circulation of the Blood.—The blood is pumped throughout the body in a system of closed tubes, the blood-vessels, which are the arteries, capillaries, and veins. It is pumped through two systems of vessels: the general, or the systemic, system, extending from the heart through all the body except the lungs, and back to the heart; and the pulmonary system, extending from the heart through the lungs, and back to the heart. If we trace a blood-corpuscle in its

journey (Fig. 12), starting at the aortic valve, *n*, we shall find that when it leaves the heart it enters the aorta, *a*, then passes into one of the many branches of the aorta, and thence through smaller and smaller arteries, until the artery becomes a capillary, in a muscle, for instance. The corpuscle passes through a fine capillary, *b*, of this muscle, and enters a small vein, *o*, whence it moves on through larger and larger veins till it finally enters the vena cava, *c*, which carries it to the right auricle, *d*, of the heart. The right auricle pumps it into the right ventricle, *e*; the right ventricle pumps it through the pulmonic artery, *p*, into the lungs, *f*. It passes through the capillaries of the lungs into the pulmonic veins, *q*, then down to the left auricle, *g*, which then pumps it into the left ventricle, *h*, and this through the aortic valve, the starting-point. If we should trace another corpuscle it might take the following course. From the left ventricle it passes into the aorta, then through the mesenteric artery, *m*, perhaps to the capillaries of the intestines, *i*, through the mesenteric vein to the portal vein, *j*, which carries it into the liver, then through the capillaries of the liver, *k*, into the hepatic vein, *l*, through this into the vena cava, to the right side of the heart,

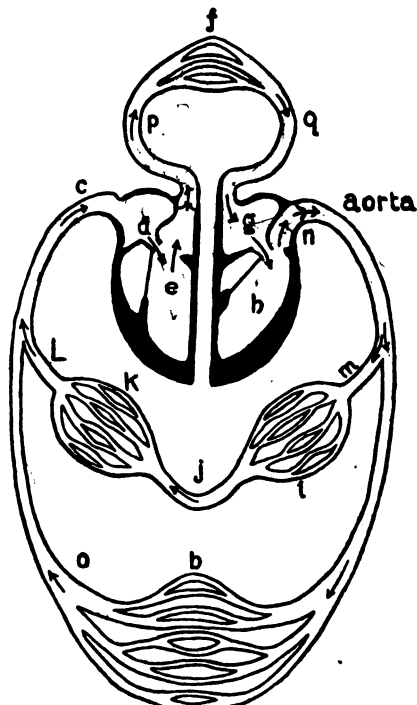


FIG. 12. Diagram of the circulation of the blood. *a*, aorta; *b*, the capillaries of the systemic circulation (*e. g.*, muscles, brain.); *c*, vena cava; *d*, right auricle; *e*, right ventricle; *f*, capillaries of the lungs; *g*, left auricle; *h*, left ventricle; *i*, capillaries of the stomach and intestine; *j*, the portal vein; *k*, capillaries of the liver; *l*, hepatic vein; *m*, mesenteric artery; *n*, aortic valve; *o*, a vein anywhere in the body except the lungs or abdominal viscera; *p*, pulmonic artery; *q*, pulmonic vein.

and then as before. In this case it passed through three sets of capillaries, those of the intestine, of the liver, and of the lungs. This latter is known as the "portal circulation."

We see, therefore, that we really have two hearts which do not directly communicate with each other (Fig. 13). The right heart, *A*, consisting of auricle and ventricle, pumps the blood through the lungs; and the left heart, *B*, auricle and ventricle, pumps the blood through the body. These two hearts are bound together into one, but their union does not imply any direct communication so far as the blood current is

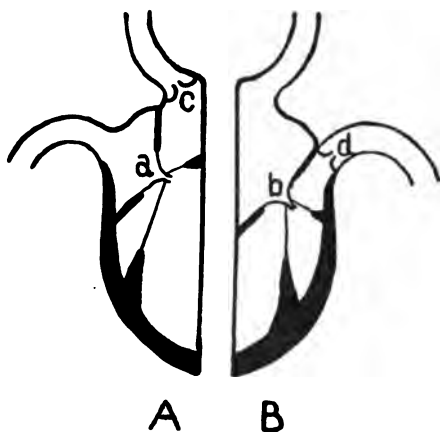


FIG. 13. Diagram of the two hearts. *A*, the right heart; *B*, the left heart. *a*, tricuspid valve; *b*, mitral valve; *c*, pulmonic valve; *d*, aortic valve.

concerned. That the blood may always be forced in the right direction, the heart is provided with valves. When the right ventricle contracts, the tricuspid valve, *a*, prevents any back-flow to the right auricle. When the contraction is over and the ventricle is limp, the pulmonic valve, *c*, prevents the blood from flowing back into the ventricle. In the same way on the right side the mitral valve, *b*, prevents the back-flow into the left auricle, and the aortic valve, *d*, prevents the back-flow into the left ventricle.

We speak of the contraction of the heart as its "systole;" the period during which it is limp and resting is called its "diastole." The valves are membranes of wonderful strength, yet almost as thin as tissue paper. The edges of the mitral

and tricuspid valves are anchored by the chordæ tendineæ, which are fine but very strong threads.

The heart is a wonderfully powerful little pump. It contracts about seventy times each minute, with each pulsation throwing forcefully about 100 c.c. of blood from each ventricle. It must by each beat throw exactly the same amount from each ventricle. If it did not, the blood would accumulate in either the pulmonary or the systemic circulation, and a difference of only one drop per beat would mean about 300 c.c. per hour. Since the right side has only to pump the blood through the lungs, its walls need not be nearly so strong as those of the left side, which has the much harder task of forcing the blood to the most distant parts of the body. The 100 c.c. of blood are expelled into the artery with such force, that, if an artery is cut the blood will spurt fifteen or twenty feet. The sudden gush of blood from the ventricle sends a "pulse wave," as we call it, down through the arteries even to the capillaries.

The little corpuscle we were following travels through the arteries at the rate of about one yard in three and a half seconds, or about one mile per hour. The capillaries are very fine, and their consequent resistance to the passage of the blood is so great, that in them it travels very slowly, only about one yard per hour; but since they are only about $\frac{1}{2}$ mm. long it takes only about one second to pass through them into the veins. Here the corpuscle travels in a steady stream instead of with jerks, as in the arteries, and faster and faster as it approaches the heart. The large veins are not nearly equal in calibre to the sum total of the smaller veins which feed them, and just as the water in the narrow part of a river flows more rapidly than it does through the meadows which that river drains, so the blood flows faster and faster as it reaches the heart. It has been reckoned that the total capillary "bed" (as one speaks of the "bed" of a river) is about eight times as wide as the aorta which feeds it.

The whole circuit of a corpuscle, from the aortic valve around the body and back to that valve, requires only about twenty-three seconds. One-fifth of this time is required for the circuit from the heart through the lungs and back again to the heart. To push this corpuscle through the whole circuit requires twenty-seven heart-beats; and, since the

heart expels about 100 c.c. of blood with each beat, every drop of blood in the body could pass through the heart in fifty-three beats, or in about forty-five seconds.

The work necessary to accomplish this task is enormous. The heart is about the size of a man's fist. It works day and night, and each hour accomplishes as much work as do the strong muscles of the legs of a man who weighs 140 lbs., and who every hour climbs a staircase sixty feet high. And yet the heart does not work all the time. We find that it rests twelve hours a day, if we add together the pauses between its beats, during each of which it is perfectly limp, and so completely tired out that at the beginning of this pause a strong electric current could not arouse it. For three hours a day it does light work, that of pumping the blood from auricles into ventricles, a relatively easy task. It works hard for nine hours a day, the sum of the moments consumed by the ventricle in forcibly pumping the blood into the arteries.

The arteries are always overfilled, and their walls are considerably stretched by the large amount of blood within them.* Since the arteries are very elastic and are distended, their elasticity is, between heart-beats, constantly forcing the blood on into the small arteries and capillaries, and keeping up a continuous circulation, although the heart-beat is periodic.

THE BLOOD-VESSELS

The vessels through which the blood circulates may be divided into three classes: the arteries, veins, and capillaries. The arteries begin at the heart with the aorta and carry the blood to all parts of the body, dividing as they go into smaller and smaller branches until they reach the capillaries. The walls of the arteries must be very strong, since the heart-beats keep the blood in them under such high pressure. Their walls (Fig. 5) consist of three layers. The inner layer is the so-called intima, *A*, a thin membrane of flat cells, which furnish a surface suitable for contact with blood circulating within the artery. Outside the intima is a thick media, *B*, which consists of muscle fibres mingled with strands of strong elastic tissue. This muscle tissue is of the so-called "involun-

* The pressure of the blood in the aorta is equal to 250 mm. of mercury, which means that it would force blood through a vertical pipe about 2 metres (nearly 6 1-2 feet) high.

tary" type; that is, we cannot control it by our will. It strikingly differs in appearance from the voluntary muscle, of our arms for instance, and differs from it also in that it can without tiring remain contracted practically for its lifetime. The media is the layer which makes the artery strong, and able to stand the high pressure within it. Outside this is a thick but rather weak layer of connective tissue, the adventitia, *C*, which binds the artery to the structures through which it passes, and serves as packing to fill the crevices around the arteries. The muscle of the media is under nervous control. When a person blushes, for instance, the nervous impulse from the brain allows the media of the blood-vessels of the cheek to relax, and hence the artery dilates—that is, its lumen becomes much larger—and much more blood rushes

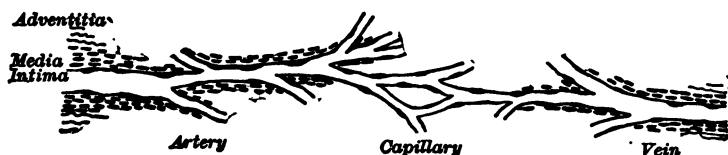


FIG. 14. Diagram of an artery, capillary and vein. Note how the walls of the artery "thin out" until the wall of the capillary is only the intima, a single layer of cells. (Of course the transitions from artery to capillary, etc., are actually very much more gradual.) Magnified 300 times.

to the capillaries. If the skin is chilled a person becomes pale, because the media contracts, and reduces the lumen, sometimes to one-half its previous size.

As the arteries (Fig. 14) by dividing get smaller and smaller, so the media and adventitia become thinner and thinner until the walls of the smallest arteries consist of the intima, and a very thin layer of muscle fibres. At this point begin the capillaries, which are about $\frac{1}{100}$ of an inch in length, and just wide enough for one red corpuscle to squeeze through. At their other end begin the smallest veins. The intima of these has around it a very thin layer of media. As the veins unite to form larger and larger veins, the media gets thicker, and then an adventitia is added; but the walls of even the largest veins are thin compared with those of an artery of the same size, since the blood within the veins is under much less pressure.

If one were asked what part of this circulation—the heart, aorta, small arteries, capillaries, veins, and, finally, the largest

vein, or vena cava—was the most important, he would be tempted at first to think of the heart and the larger vessels; but in fact the most important part of the whole system is the tiny capillary, which is only about $\frac{1}{4}$ of an inch in length. It is in order that the blood may pass through these tiny tubes, the capillaries, that this whole “cardio-vascular system” exists.

The function of the blood is to nourish the body. The nourishment within the blood-vessels cannot get through the walls of the arteries and veins, but can get through the single layer of intima cells, *a*, of the capillaries (Fig. 15). In these tiny vessels the blood flows very slowly; the plasma soaks through their walls, furnishing new food to the tissue-cells, *b*,

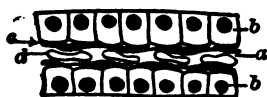


FIG. 15. A capillary and the cells which it furnishes with food and oxygen and whose excreta it removes. *b*, tissue cells; *a*, lymph space between cells and capillary wall. *d*, red blood-cells represented as squeezing through the narrow capillary. (Magnified 600 times.)

and taking up much of the waste matter which these cells have excreted. It is here that the oxygen leaves the red blood-cells and goes to the tissues, and the carbon dioxide leaves the cells and goes to the plasma. The importance of the capillary may become clearer if one thinks of the stations of the railroads which pass through Baltimore.

Suppose there were no stations, and that the inhabitants of Baltimore were starving; that freight-trains full of food, enough to furnish the citizens in great abundance, were thundering through the city on the main tracks every ten minutes. The inhabitants of Baltimore would continue to starve unless these trains should stop and the food should be unloaded so that the citizens could get it. So, the heart might pump the blood through the arteries into the veins and back to the heart in abundant volume, but the tissue would starve if it were not possible for the food to be unloaded in the capillaries, the stations.

In most of the body we have also lymphatic vessels, which drain the tissue spaces (Fig. 15, *c*) of their waste matter and make it unnecessary for this to flow back into the capillaries. These lymphatics, as they are called, by uniting form larger and larger vessels, pass through lymph-glands, and then combine in the large “thoracic duct,” which empties into the large vein on the left side of the neck. But it is a question of

how much use these lymph-vessels are in the ordinary life of the cells. If the tissue becomes diseased they are very important, but the most of the contents of the thoracic duct is food absorbed from the intestinal wall.

THROMBOSIS.—A thrombus is a blood-clot which forms inside a blood-vessel. The vessel is then said to be *thrombosed*, and the condition is called *thrombosis*. It may be a small clot clinging to the vessel's wall, or it may completely plug the vessel (Fig. 16), in which case it prevents the blood from passing any further along that course. These clots consist of fibrin and entangled blood-cells and strongly resemble the clots mentioned on page 11. We think that either there must be an injury to the vessel wall or germs must have settled and started an inflammation of the wall before the blood could clot at that point; for the normal intima should not allow this to occur.

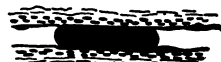


FIG. 16. A small artery plugged by a thrombus.

FIG. 18.

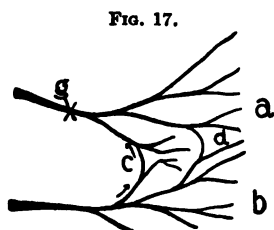
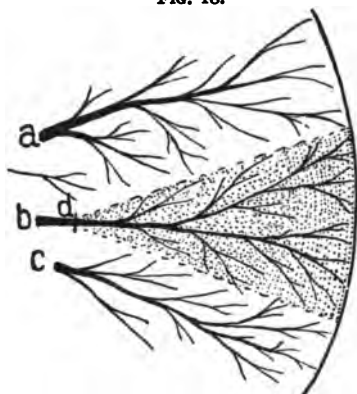


FIG. 17.

FIG. 17. Two anastomosing arteries. *a*, and *b*, are two small arteries which are connected by two small communicating branches or "anastomoses," *c*, and *d*. If *a* should become plugged at *g*, the circulation through *a* would not stop since the blood would flow from *b* through *c* and *d* into the branches of *a*.

FIG. 18. End-arteries, and an infarct. *a*, *b*, and *c* are three small arteries which have no communicating branches. If *b* becomes plugged at *d* then all the tissue supplied with nourishment by *b* must die. This dead area is an "infarct." (The dotted triangle.)

The results of a thrombus will depend entirely on the nature of the circulation where it occurs. If it occludes, or plugs up, a vein the blood will be dammed back, but the blood can reach the heart along some other channel. There are two kinds of arteries in the body—those which "anastomose" (Fig. 17), that is, send branches across to neighboring arteries; and those which do not. The latter are termed "end-arteries"

(Fig. 18). If an end-artery, *b*, gets plugged, as at *d*, all circulation along that channel stops, and the tissue which that artery feeds must die. If it is not an end-artery (Fig. 19), then the plug, *y*, may make little trouble, for the arteries in the neighborhood, *b*, which are not plugged will send enough blood through these short communicating or "anastomosing"

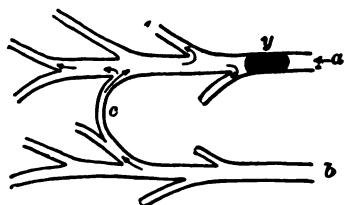


FIG. 19. Anastomosing arteries. *a* and *b* are connected by an anastomotic branch *c*. If *a* is plugged by the thrombus *y* the circulation in *a* will be maintained by *b*.

branches, *c*, to fill the artery beyond the plug and so keep up its circulation. Hence the tissue which the plugged artery feeds will receive sufficient nourishment. The small anastomosing arteries become larger and larger until they are able to carry all the blood necessary—that is, until a sufficient collateral circulation is developed.

There are practically no end-veins, and the anastomosing branches are so numerous that to establish a sufficient collateral venous circulation is easy; hence a venous thrombus does relatively little damage. It is a very interesting fact, and one hard to explain, that the most important organs of our body—the heart, brains, kidneys, spleen, intestine, *e.g.*—have end-arteries, and if any branch of an artery in them becomes occluded a certain amount of tissue must die. Since the arteries as a rule flow from the centre to the periphery of an organ, and branch like a tree, it follows that the area which dies will be wedge-shaped, with its base on the surface of the organ. These dead areas are known as "infarcts" (Fig. 18).

EMBOLISM.—An embolus is a thrombus which has become dislodged—that is, which breaks loose and is swept on in the blood current through the blood-vessels until it comes to one which it cannot pass through, and which it plugs tightly (Fig. 20). This process is called *embolism*. The majority of such thrombi form in the cavities of the heart, and especially on the edges of the heart valve. An embolus from a vein or a cavity

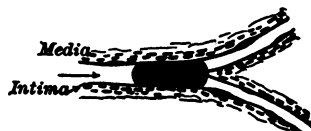


FIG. 20. An embolus plugging a small artery. The embolus *a* was forced along by the blood stream until it reached a bifurcation of the vessel; here it stops.

of the right heart will travel to the lungs, and occlude a branch of the pulmonary artery. One from the pulmonic veins, or a cavity of the left heart, or a large artery, will plug a small artery. Where the embolus stops it becomes again a thrombus. Its results depend on the artery in which it happens to lodge. If this is an artery with sufficient anastomosing branches, there is no result, but if it is an end-artery, then the result will be the death of all the tissue which normally receives its nourishment from that artery. It is an interesting and an inexplicable fact that those organs in which an embolus can do the most damage are the very ones in which the embolus is most likely to lodge. It often goes to the heart muscle through the coronary artery which leaves the aorta just behind the aortic valve. The result of this case is the sudden death of a portion of the heart wall and, unless this be a very small portion, the paralysis of the whole heart. Another favorite spot for an embolus to strike is the brain, since the vessels to this form the straightest path for an embolus leaving the heart; and, unfortunately, of the whole brain that portion most often hit is the one which ought to be most carefully guarded, the internal capsule of the left hemisphere. If one should try to cripple the whole of the telephone system of one section of a city, he might go about it in two ways. He might pass through the streets cutting the wires wherever he found them; but this would take him a long time. He could go to the central station and there with one blow sever every wire at the point where they are all bound together in one bundle as they leave the station before scattering over several square miles. So it is in the brain. The fibres leave the wide area of the cortex. They collect together in one small bundle at the base of the brain, known as the internal capsule, pass into the spinal cord, and are all distributed to the whole of the opposite side of the body, except a few which go to the other side. Hence it is that a small embolus in the internal capsule can paralyze almost every muscle on the opposite side of the body. Our speech is controlled from the left side of the brain, and from an area adjoining the "motor area;" and hence an embolus to this most vulnerable point deprives a person of speech as well as of the use of the right side of his body. It is hard to understand why this most important point should be the most vulnerable for an embolus.

The spleen and the intestine are also favorite places for the embolus to strike. In the case of the intestine, if the embolus is at all large, death is very certain to follow. Emboli to the kidney or spleen always produce infarcts, but not with so serious results. If, however, the embolus contains germs, then wherever that embolus lodges will be a centre of infection and the starting-point perhaps of an abscess.

ARTERIOSCLEROSIS.—The word implies “hardening of the arteries.” This is one of the most important diseases to consider, since it indirectly kills the majority of men. The media is the most important layer of the vessel wall, and being the most important layer is the most sensitive to injury.

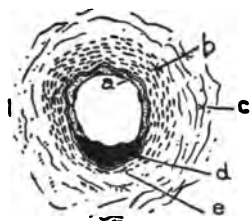


FIG. 21. Arteriosclerosis of a small artery. a, intima; b, media, c, adventitia; d, a mass of thickened intima which strengthens the weak spot e of the media.

Any agent which attacks an artery—a poison, for instance—will injure this layer most. Suppose that in a small area of the artery's wall some muscle fibres should be killed by a poison (Fig. 21, e). At that point the media would become thin and weak. One of two things might happen: either the wall would burst at that point, because of high pressure of the blood current within, as happens in a water-main; or, since the wall is elastic, it would “balloon out,”

forming a blood tumor, or “aneurism.” The body protects itself against such accidents by increasing the thickness of the intima at any weak point, d, and hence as the media gets weaker the intima gets stronger, and a small, thick plaque of intima, d, forms here. But this plaque is nothing but ordinary weak scar tissue which has very little vitality, is poorly nourished and itself soon dies. Lime salts are then deposited in the dead tissue, and a small fragment of hard shell is there formed; or this dead tissue will slough out and leave an ulcer in the arterial wall. This plaque-formation is known as arteriosclerosis. Sometimes the arteries are thickened along their whole course, sometimes only in certain areas, and sometimes in little spots over wide areas. If an artery has a large number of these plaques along its course it is by no means a normal artery. Also, these plaques choose as a favorite point for their formation the spot where a small branch artery leaves

an artery (Fig. 22, *a*), and hence this branch has its mouth partially choked by the plaque of sclerotic tissue.

The results of arteriosclerosis are easy to understand. Sclerotic arteries are more or less rigid, not elastic, tubes, and so more force is required to pump blood through them. The first result, therefore, is that the heart must work much harder in order to keep up sufficient circulation. We know that if we exercise any of our muscles they get thicker and stronger. So it is with the heart. Increase its work, and it enlarges, or "hypertrophies." But, although larger and stronger, it is not a better heart than normal; in fact in some ways it is a less serviceable organ, as we shall see later. The next result of arteriosclerosis may be stated as a law in pathology: that whenever a tissue-cell receives insufficient food it gets weaker or dies, and if it dies its place is taken by a tissue requiring less food. That tissue which requires least food, and which is quite generally used to replace starved cells, is fibrous or scar tissue, a tissue which cannot perform the specific functions of the cells which it replaces. Those organs which perform the most important functions are built of cells which require the most food, and which are most sensitive to any reduction in this food supply. To illustrate: a person with general arteriosclerosis may have heart walls in which much heart muscle fibre is replaced by inert scar tissue, the result of arteriosclerosis of the coronary arteries, the vessels which supply the heart wall with blood. The heart in this case is called upon to increase in strength because of the arteriosclerosis of the whole body, but since this disease is also in its own walls it is very hard for it to respond. A kidney fed by sclerotic vessels may be about one-half its normal size; a liver may consist of about one-half scar tissue; and in the brain may form areas of atrophy, weak points at which later a hemorrhage is likely to occur.

The causes of abnormal degrees of arteriosclerosis may be reduced to heredity, wear and tear on the arteries, and poisons; and the degree of the sclerosis will depend on the quality of the material of which the vessels were originally built. The most common cause of all, of course, is advancing age, for



FIG. 22. Arteriosclerosis. A small patch of arteriosclerosis *a* is partially occluding the mouth of a small branch artery.

sclerotic arteries are almost normal for elderly persons. But there are some families whose members have arteries which grow old too soon. These persons were born with "poor rubber in their tubes," and hence their blood-vessels, and so their whole bodies, show age more rapidly than do members of other families. Since the health of the whole body depends to a considerable degree on the vascular system, there is much truth in the old adage that a "man is as old as his arteries;" that is, some persons at forty have arteries as thick as those of most men at seventy, and can claim little better health than can the latter.

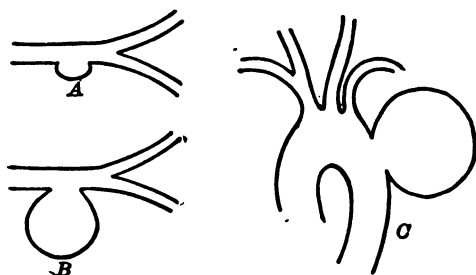


FIG. 23. Sacular aneurisms. A, a small sacular aneurism; B, a later stage of A. C, sacular aneurism of the arch of the aorta. A very common and a huge form. These may be several inches in diameter.

Excessive wear and tear is one of the most important causes of early sclerosis, and this is due to abnormally high blood pressure. This is the reason why persons who do especially hard muscular work have arteriosclerosis; also, why those who habitually overeat, and especially those who worry a great deal are subject to it. Worry is now one of the most potent causes, and explains why so very few of our stock-brokers and business men who work under high nervous tension reach old age. Under this same heading probably come cases of Bright's disease, for their blood pressure is abnormally high. A great many cases are due to poisons such as alcohol or lead, also to the poison of certain fevers, as gout, or typhoid fever.

The treatment of arteriosclerosis is very important since, as we have said, arteriosclerosis indirectly kills the majority of men. The damage already done to an artery cannot be repaired, but the process can be prevented from getting much

worse by stopping the cause—the alcohol, or the cause of a high blood pressure, etc. Persons with sclerotic arteries should live very quiet lives, avoiding excesses of all kinds. They should keep their bowels open by proper medicines, and the skin in excellent condition by frequent baths. They should studiously avoid alcohol in all forms and should restrict themselves to a very limited diet. Of drugs a very useful one is potassium iodide. This is given in an almost saturated solution, one drop of which contains one grain of the salt. It is best taken in milk after a meal. For these cases about 10 grains is the proper dose, which should be taken three times a day. Nitroglycerin is sometimes used, but its value is rather doubtful. Of this we give one drop of a 1 per cent. solution three times a day as the first dose, then increase the dose if necessary, stopping when a headache and throbbing in the head warns us to stop.

ANEURISM.—As we have already learned, the arteries are very elastic tubes, through which flows a fluid under high pressure. If the wall gets weak at any point and does not burst, and the weak spot is not repaired by the strengthening of some other coat, as the intima, the wall will be blown out, very much as a rubber bag is inflated, by the pressure of the blood within, and a large pulsating blood tumor be formed. This is called an aneurism. If the weak spot was very limited in extent, so that the aneurism projects from one side of the artery, we have a “saccular aneurism” (Fig. 23, A, B, C); if the whole artery was dilated, we have a fusiform aneurism (Fig. 24, A, B). The body at once protects itself by forming new tissue around this tumor, which in this way soon gains a thick wall. But the tumor may rupture and the blood work its way along between other organs, scar tissue meanwhile forming a wall around the tumor to limit the escaping blood. This is known as a “dissecting aneurism.”

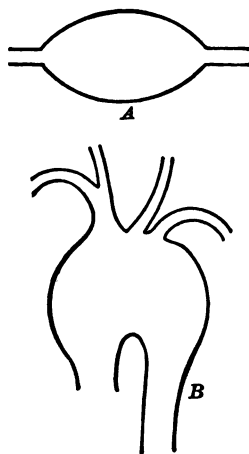


FIG. 24. Fusiform aneurisms. A, a fusiform aneurism of a small artery. B, fusiform aneurism of the arch of the aorta, the commonest form. This is also called “diffuse aneurism,” or “dilatation of the arch.”

The symptoms of aneurism are those of tumor, plus the pulsation due to the pulse wave of the blood inside.

An aneurism may rupture externally or into a cavity of the body, and the person bleed to death in a very few minutes; or the blood within the sac may clot solidly. If the latter occurs the aneurism is cured. We have then merely the pressure symptoms of a solid tumor. This clotting is, of course, what we try to accomplish in treatment. The patient is put to bed and kept as completely at rest as possible in order to reduce the number of heart-beats even fifteen per minute, and thus in some degree to quiet the blood within the tumor. He is also almost starved that his blood pressure may be low. In this way nearly five of every twenty-four hours' work is spared the heart. Or, about ten feet of wire are inserted into the tumor and an electric current run in, in the hope that the foreign body and the electric current will stimulate coagulation. This succeeds with some saccular aneurisms. The aneurisms of small arteries are more easily cured by partially or wholly occluding the vessels, but since most aneurisms are on the aorta or its big branches this is rather a difficult proposition.

Large aneurisms are not very common, but minute aneurisms are exceedingly so. For instance, a hemorrhage from the lungs in tuberculosis, when it occurs late in the disease, is nearly always due to the rupture of a minute aneurism in the wall of an exposed pulmonary artery. The same is true of a hemorrhage from the stomach in gastric ulcer, and hemorrhage into the brain, *i.e.*, "apoplexy." In nearly all of these cases the artery was injured first at one point, and there developed a tiny saccular aneurism which is the weak point which later ruptures.

CHAPTER II

DISEASES OF THE HEART

Endocarditis.—Endocarditis means inflammation of the endocardium, the membrane which lines the cavities inside the heart. It is practically the same as the intima of the blood-vessels. Inflammation of the endocardium is usually limited to small patches. On these inflamed spots clots, or thrombi, of fibrine form, here called “vegetations,” because

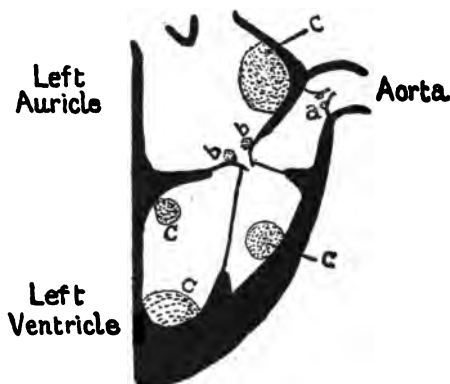


FIG. 25. The vegetations of acute endocarditis in their most common locations. *a*, vegetations on the aortic valve (ventricular surface of flaps); *b*, on the mitral valve (auricular surface of flaps); *c*, mural thrombi.

these thrombi project from the endocardium and in shape somewhat resemble the cauliflower. The inflammation is due to germs which are brought in by the blood stream and deposited here.

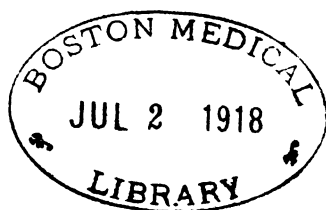
Any part of the endocardium may be thus diseased. When a clot forms on the wall of one of the cavities of the heart it is called a “mural thrombus” (Fig. 25, *c*). Such clots, are, as a rule, rather large,—about the size of a marble. They are usually several in number and when numerous may almost fill an auricle or the tip of a ventricle. These clots make very little trouble for the patient so long as they remain attached,

but when one breaks loose it is swept along with the blood current into the arteries as an embolus, and the result will depend on the vessel in which it finally lodges.

But, unfortunately, endocarditis attacks oftenest the valve flaps themselves, and of these important structures the place where it can do the most harm,—that is, the edges of the valve flaps, the margins which are pressed together when the valve is closed (Fig. 25, *a*, *b*). The germs are deposited on the edges, probably because these, being those parts of the endocardium on which is the greatest strain, are, therefore, most vulnerable. A row of these small vegetations along the margins of a valve flap is the chief cause of valvular heart disease.

ACUTE ENDOCARDITIS.—There are two kinds of acute endocarditis, the simple and the ulcerative. Although the difference is one of degree only, yet it is important. This difference depends on the virulence of the germ causing the inflammation. In “simple” endocarditis the vegetations are like tiny translucent beads, about the size of the head of a pin, and are often overlooked, or seen only by holding the flaps up to the light. The endocardium around them is a little red. These innocent-looking little clots seem harmless enough, and are so in themselves. They seldom while fresh cause any local symptoms, but the late effects of even the simple vegetations are often very important. The small clots may disappear in a short time and leave no trace of a vegetation. In health these valve flaps are marvellously strong membranes, and yet almost as thin as tissue paper. Sometimes, perhaps, this valve never suffers any further consequence, but the rule seems to be that this simple endocarditis sets up a slow, insidious thickening of the flap, a process which may continue for years. The flap may look almost normal, but when examined carefully it is seen to be just a little shorter, just a little thicker, than normal, and is just a little shrivelled along its edges—not much, but enough to prevent that flap from perfectly closing the orifice of the valve, and hence to allow a leak.

In “malignant” or “ulcerative” endocarditis the clots are larger, even the size of the end of a finger, and some are quite an inch long. They are sometimes firm, opaque, yellow clots of fibrine, and hang loosely attached to the valve by a slender pedicle. They flap in the blood current, and often



break loose and travel as emboli. Sometimes many form and break loose, each one plugging some blood-vessel; and then it seems as if the heart were bombarding the body. But these emboli may do more than merely plug a vessel, for they are sometimes full of the bacteria which caused their formation. Wherever these settle they become a centre, or "focus," of infection; that is, an abscess may form around each clot. The clot when torn away leaves an ulcer behind. An actual hole may perforate the valve flap. The spot on the valve where the clot formed is much weakened by the inflammation, and the pressure of the blood current may puff it out into an aneurism. The immediate result, therefore, may be a flap with a torn, frayed, weak edge, with chordæ tendineæ broken or torn loose. In fact practically nothing of the valve may be left but a row of tags and tatters which are of no use in closing the orifice. Between these two extremes (a slightly thickened or a torn flap) are all degrees of injury to the flaps. The clots may calcify and feel like masses of shell. This severe, malignant endocarditis seems to affect especially valves which are the seat of older simple endocarditis.

We have spoken only of valve lesions causing a leak, or valvular insufficiency. There is another possibility. The result of the endocarditis may be partial closure of the valve orifice. The inflamed flaps may grow together along their margins, or, by contracting, narrow the orifice of the valves, or become very stiff and protrude into the orifice. This result is called "stenosis" of a valve. A stenotic-valve orifice is smaller than normal, but although smaller it is usually also insufficient since the stiff valves do not close the narrow orifice perfectly.

So far as we know, acute endocarditis is always due to some germ. We know the germ is carried there by the blood, but the problem is to find out whence comes this germ—that is, what is "the portal of entry." Acute endocarditis often follows so-called "blood-poisoning;" that is, through a prick by a dirty nail or pin, very poisonous germs get into the blood. Here they multiply and are carried over the whole body, settling at various points, and where they settle they start up new centres of infection. The heart valves are favorite places for these germs to settle. Simple endocarditis most often follows acute rheumatic fever, and many think that the

germ which causes both the joint and heart inflammations enters the body through inflamed tonsils. St. Vitus's dance also, often associated with rheumatism, is in many cases followed by acute endocarditis. Scarlet fever and diphtheria are frequently followed by heart disease. In fact, the germ of any acute infectious disease may affect the heart. Pneumonia is apt to cause a severe ulcerative form; while gonorrhoea causes the very worst form, with the largest vegetations.

In very young children the valves of the right heart are more often affected; in older children and adults, those of the left. Of all valves the mitral is most often affected, and the aortic next.

During an attack of simple endocarditis there may be practically no symptoms at all, while those of a severe case of the acute ulcerative form may be very obscure. Unless the heart is examined the nature of the disease may be entirely overlooked. There is sometimes a fever which lasts for weeks or months. Some cases closely simulate malaria; others, abscesses of internal organs.

If a little blood is taken from a vein the germ can often be found.

The treatment of endocarditis seems hopeless, but it is not. Many of these cases could have been prevented, had the throat been carefully treated. Cases of acute fever (pneumonia, *e.g.*) can be kept very quiet, and hence complications may be warded off. This is particularly true of rheumatism, for children with slightly swollen joints are often allowed to run about much too early. The patient should rest perfectly quietly in bed while the endocarditis is acute, and for a long time afterwards, in order that the heart may have plenty of time to get used to the new conditions—that is, to the leak with which it must work. This may mean rest in bed for months after the patient feels well, and the rest should be of the mind as well as of the body. During this time an ice-bag is kept over the heart, a few blisters at the apex, and for a long time the patient is given potassium iodide. A patient with a slight leak in the valve can, by leading a quiet life, live years—perhaps an average lifetime—without suffering any especial symptoms. He may, indeed, never discover that he has any heart trouble unless examined by a life-insurance or army examiner. But some unusual strain or severe

exertion may overtax the heart, and death may be the first sign of disease. In other cases a sudden strain is followed by months or years of invalidism. A patient is fortunate if he discovers his trouble before he "strains" his heart; for by avoiding physical, mental, and nervous fatigue, and by avoiding colds and gastro-intestinal upsets, he may never suffer from his trouble.

Endocarditis is called acute or subacute as long as active inflammation is present.

CHRONIC ENDOCARDITIS is the result of the acute—it is the resulting permanent injury to the heart. But there is another form of chronic endocarditis, affecting especially the aortic valves, which is very important. We mean the "arterio-sclerotic form;" for the same process which causes thickening of the intima of the aorta can creep onto the aortic valves—which are really but folds in the intima, called here the endocardium—and produce slight thickening, stiffness of the flaps, and slight curling of their edges—not much, but just enough to prevent a perfect closure of the aortic orifice.

The Mechanism of Valvular Heart Disease.—We have two hearts (Fig. 13), a right, *A*, and a left, *B*, each with an auricle and a ventricle. These two hearts have no direct communication with each other. The right heart pumps the blood through the lungs to the left heart—the "lesser" circulation—while the left heart pumps it through the body and around to the right heart—the greater or "systemic" circulation. Both auricles contract at the same time, the right forcing the blood through the tricuspid valve, *a*, into the right ventricle, and the left through the mitral valve, *b*, into the left ventricle. The ventricles when full contract forcibly. This pressure in the ventricles first closes the tricuspid and mitral valves, then when on the right it is greater than its pressure in the pulmonic artery, and on the left is greater than that in the aorta, it forces these two valves, the pulmonic, *c*, and aortic, *d*, open, and blood is forced into these two arteries. Each beat forces about one hundred cubic centimetres of blood out of each ventricle. As soon as the ventricles are empty their walls become perfectly limp. The blood in both pulmonary artery and aorta is under considerable pressure and tries to rush back into the ventricles, but is checked by the pulmonic, *c*, and aortic, *d*, valves.

When the ventricles contract and close the mitral and tricuspid valves, the muscle walls and the valves together make a sound which is best described by the syllable "lub." When the pulmonic and aortic valves close they make a shorter, sharper sound, like the syllable "dub." The heart sounds produced by one contraction or "systole" are thus represented by the sounds "lub dub." Graphically they may be represented by Figure 26. The interval from the



FIG. 26. A graphic representation of the normal heart sounds. *a*, the first sound; or "lub"; *b*, the second sound or "dub." *c*, one heart beat, of which cycle "*S*" is the systole (from the beginning of the first to the beginning of the second sounds), *D*, the diastole (from the beginning of the second to the beginning of the next first sound).

beginning of the first sound to the beginning of the second is called "systole." That from the beginning of the second to the beginning of the next first, "diastole."

The heart occupies a position indicated in Figs. 48 and 51. Usually one can see the apex of the left ventricle move the chest wall with each beat at Fig. 51, *A*, a point just below and median to the left nipple in the fourth or fifth interspace. This spot is called the "point of maximum impulse," or the P. M. I.,



FIG. 27. A diagram of the pulse waves. (A modified "pulse tracing.") *a*, the ascent of the wave; *b*, the descent, and *c*, the diastolic wave.

and this pulsation is called the "apex beat." The areas where the various valve sounds are best heard are here indicated. Thus *A*, is called the "mitral area." The mitral valve is at some distance from *A*, but its sounds are best heard at that point; *B*, is the pulmonic area, *C*, the aortic, and *D*, the tricuspid area.

This sudden addition of about one hundred cubic centimetres of blood to the contents of an artery already full stretches its walls still more, and starts a wave which travels down the arteries to the capillaries, and which is called the

"pulse wave" or the "pulse." If this wave is represented graphically, as by Fig. 27, its ascent, *a*, due to the sudden influx of blood, is sharp; its descent, *b*, is slower, since the blood is slowly squeezed onward by the overdistended, elastic arteries into the smaller vessels. On this descent is seen a small wave, *c*, called the "dicrotic wave," which is produced by the snap of the aortic valve as it closes. Normally the pulse is very regular, the beats following each other with great regularity. Even in severe aortic-valve disease the rhythm is regular, but in mitral-valve disease, and especially in myocarditis, the irregularity is a marked feature of the case.

A valve which leaks is said to be "insufficient;" the condition is called "valvular insufficiency" or "valvular incompetence." When the orifice is too small, "stenosis" is said to exist. If the valve is so torn or shrunk or stiffened that it cannot fill a valve orifice of the normal size, the condition is "absolute insufficiency." "Relative insufficiency" is a different condition. The heart is an elastic, hollow, muscle bag (Fig. 28), and its capacity is dependent on the "tone" of its walls. When extra strain occurs and the walls are unable to hold their tone, or degree of contraction, they "stretch," and the volume of their chambers is increased. But the size of the orifices in these chambers will increase also. The flaps, *c*, of a normal valve are of just sufficient size to fill an orifice of normal size, and they cannot stretch; hence, when the orifice is larger than normal the flaps cannot close it, that is, the valve is relatively insufficient.

The normal heart uses, under ordinary circumstances, but a small portion of its strength for each beat; the rest is held in reserve. But let a normal person run violently or lift some heavy weight, and the heart must fall back on its reserve strength. If the total strength of the heart is represented by the line *a-b* (Fig. 29, *A*), under ordinary circumstances *b-c*

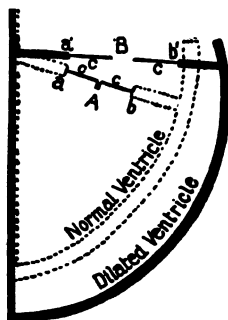


FIG. 28. A diagram of a normal and a dilated ventricle. When because of weakness the muscle wall stretches, and the cavity of the ventricle "dilates," the orifices will dilate also. The valve flaps *c*, can close tightly the orifice *a-b*, but cannot close the orifice *a'-b'*, and yet there has been no change in the flaps *c*. The valve *A* is "competent," but *B* is relatively "incompetent," or "insufficient" (ly closed).

will represent the force necessary for each beat, and $a-c$, the force held in reserve, which it can use when subjected to extra strain. A runner is said not to run a successful race unless he faints just when he crosses the tape—that is, unless at that moment his heart has reached the limit of its reserve strength.

AORTIC STENOSIS.—We consider this rather rare lesion first, since it presents the simplest mechanical problems. In this disease the flaps of the aortic valve have become glued

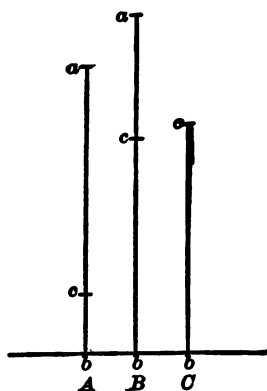


FIG. 29. Diagram of cardiac force. *A*, normal heart. *B*, heart hypertrophied because of a leaking. *C*, heart with "broken compensation." $a-b$, total heart force; $a-c$, reserve heart force; $b-c$, force used in each contraction.

together, or have become so stiffened by masses of exudate, or even calcareous plates, that they project into the lumen and partially occlude it (Fig. 30, *B*). The orifice of the valve is, therefore smaller than normal, and the left ventricle meets with obstruction when it tries to force the blood into the aorta. The ventricle must contract more slowly and with greater force than normal. The first sound will, therefore, be long-drawn, rough, and vibrating, in character—"r-r-r-r-rub-dub."

If one puts his hand over the aortic area he feels a vibration like the purring of a cat, caused by this forcible contraction's squeezing the blood through a small hole.

The pulse will be rather slow, for the heart must make slow, laborious contractions; the ascent of the wave is slow, because the blood is squeezed slowly into the aorta; and the apex of each wave is rounded (Fig. 31).

The left ventricle alone in this case has a harder task than normal to perform at each beat. Its walls will be stretched a little at first, it will use more of its reserve force, and then it will do what all muscles do when given increased work—it will become stronger and thicker; that is, it will hypertrophy. This condition may be represented by the line *B*, of Fig. 29. Its total strength, $a-b$, is greater than normal; but since its task at each beat, $b-c$, is harder, the margin, $a-c$, is much less. The man easily finds this out when he undertakes any unusual exertion.

In the course of time the heart gains strength enough to enable it to do its usual amount of work; that is, the lesion is "compensated," and the heart is in a state of "compensation."

In aortic stenosis the trouble is limited to the aortic valve, and the extra strain affects only the left ventricle. The rest of the heart would seem to be undisturbed and the circulation to progress without any changes at all. Now, suppose the lesion at the aortic valve becomes very extreme, or because of some unwise effort on the part of the patient the



FIG. 30. The aortic valve. *A*, normal valve. *B*, the flaps of the valve are so thickened by vegetations that the orifice of the aorta is partially occluded, "aortic stenosis." *C*, the valve flaps are so torn that they cannot close the orifice, "aortic insufficiency."

heart is "strained" and has no reserve left, then the left ventricle cannot throw out its hundred cubic centimetres of blood at each beat, and a "break in compensation" results; that is, *a-c*, has disappeared, and all the available energy is used to accomplish each beat (Fig. 29, *C*). The left ventricle is filled each time by the auricle with the usual amount of blood, which it can with difficulty expel. It becomes over-distended; that is, the heart—a hollow muscle bag—gets stretched, the orifices into it become larger, and soon the



FIG. 31. A pulse tracing of a case of aortic stenosis. (Compare with Fig. 27.)

mitral-valve flaps, although normal, will be unable to close the unusually large mitral orifice. Then all the results of a mitral insufficiency will follow. During the state of compensation the patient may have had no hint that his heart was at all abnormal. He may have noticed that he was not quite as good a runner as formerly, but that may have been all. When the heart reaches the state of non-compensation, however, all the symptoms of dilated heart will follow, and these are severe enough.

AORTIC INSUFFICIENCY.—When the aortic valves are so injured that they cannot quite close the aortic orifice (Fig. 30, *C*)

then the blood, always under high pressure in the aorta, will hiss back into the left ventricle during diastole; that is, between beats—when the heart is relaxed. One must remember that the aorta, and the arteries form a very elastic tree kept overfilled by the heart-beats. The elasticity of this over-distended arterial tree is always forcing the blood on into the smaller vessels, or, if not prevented by the aortic valve, back into the heart. There will, therefore, be a backward flow of blood into the ventricle, of an amount varying with the size of the leak. If all should leak back circulation would stop. The heart overcomes this difficulty much as a man climbs a slippery hill. If for every upward step of two feet in length he should slip back two feet, he would make no progress; he will try to take steps perhaps three feet long, that each step



FIG. 32. A pulse tracing of a case of aortic insufficiency. (Compare with Fig. 27.)

may leave him at least one foot higher. So the heart will try to throw out into the aorta so much blood—both that which the auricle provides and that which has leaked back since the last beat—that, notwithstanding the back-flow, the usual amount will remain in the aorta.

The ventricle throws this large amount of blood into the aorta with such force that the wave can be seen even in the smallest arteries, while the large arteries seem really to jump. As might be expected, the pulse wave when charted has a sudden high ascent, a sharp apex, and a quick fall (Fig. 32).

Because of this extra amount of blood to be thrown out by each beat the heart dilates, and, since it takes more muscular effort to accomplish each beat, the left ventricle hypertrophies. If the dilatation is not extreme enough to stretch the mitral orifice, the increased strength of the left ventricle will compensate for the whole trouble, and the circulation continue normally. But, really, the whole heart will hypertrophy somewhat, and hearts with this lesion are the largest hearts seen in any disease. The patient may for years be unconscious that he has any heart trouble at all; or he may complain of palpitation on exertion, or perhaps of dizziness on sudden movements; or he may discover the trouble only by accident. On examination, however, his heart

will be found larger than normal, the hiss of the backward flow may be heard (Fig. 33), and the character of the pulse will be quite unmistakable.

There are two groups of cases of aortic insufficiency. The first is the endocarditic group. Here an acute endocarditis has injured the flaps. Such a heart is able to compensate well even for an extreme lesion. The second is the arteriosclerotic group. This is a different matter. The flaps of the valves



FIG. 33. A graphic representation of the heart sounds in a case of aortic insufficiency. The second sound *b*, is replaced by a "diastolic blow." (Compare with Fig. 26.)

are really folds of intima, and the same arteriosclerotic changes of the aorta will creep upon, thicken, and shorten the valve flaps. But compensation here is not so easy, for the coronary arteries, which provide the heart muscle with blood, open from the aorta just behind the aortic valve.

The arteriosclerosis will narrow the mouths of these arteries and creep along their course. In order to hypertrophy, a muscle must have increased food, but here the source of

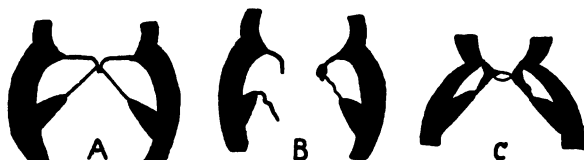


FIG. 34. The mitral valve. *A*, normal valve. *B*, the valve flaps are shrunk and torn so that they cannot close the orifice,—"mitral insufficiency." *C*, the valve flaps are so thickened that the orifice is partially occluded. The small circle represents the maximum orifice in some extreme cases,—"mitral stenosis."

supply is impaired by the disease of the coronary artery; and so the arteriosclerosis not only causes the valvular lesion but hinders the hypertrophy necessary to compensate for it.

MITRAL INSUFFICIENCY.—The mitral valve is the valve most often attacked by endocarditis. We will suppose that a little curling, shortening, fraying, tearing, or stiffening of one or both of the mitral-valve flaps (Fig. 34, *B*) prevents a perfect closure of the valve orifice during systole—that is, while the powerful ventricle is forcing the blood into the already overfilled aorta. Then the ventricle will force some

blood also back into the left auricle, and a less amount than normal into the aorta. During systole the left auricle should receive blood from the pulmonary veins alone, blood which it will force into the ventricle at its next beat. But now during systole the auricle gets blood from two directions, from the lungs and from the ventricle. It must, therefore, dilate, and, since it has extra work to do, it must hypertrophy. But this back-flow from the ventricle will also check the current of blood from the lungs, damming it back upon the right ven-



FIG. 35. Pulse tracing of a case of mitral disease. (Compare with Fig. 27.)

tricle, which must keep up the pulmonary circulation against this, a new obstacle. The result is that even a slight mitral leak will lead also to dilatation and hypertrophy of the right ventricle, and the pulmonary blood-vessels will be constantly somewhat overfilled by blood under a somewhat abnormally high pressure. The patient will, therefore, be subject to attacks of bronchitis. This cough and shortness of breath on exertion may for years be his only symptoms, even though his heart is much hypertrophied; but his face usually looks

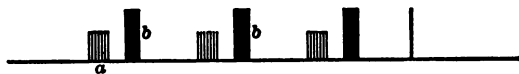


FIG. 36. A graphic representation of the heart sounds in a case of mitral insufficiency. The first sound is replaced by a "systolic blow." (Compare with Fig. 26.)

suffused, and the lips a trifle blue, while the high pulmonary pressure may cause small or even large hemorrhages from the lungs. The pulse will be small, but, what is more important, in severe cases very irregular (Fig. 35). The first sound will be accompanied or even replaced by a systolic blow caused by the blood rushing back into the auricle (Fig. 36).

MITRAL STENOSIS is a very common trouble, especially in women. The endocarditis of the mitral-valve flaps has resulted in an abnormally narrow mitral-valve orifice (Fig. 34, C). Normally one should be able to put with ease three fingers through this orifice, but in a well marked case of stenosis one can sometimes hardly force a lead pencil through

it. The valve flaps have grown together; the cordæ tendinæ have become much shortened, pulling the flap edges almost down to the tips of the papillary muscles. Or masses of calcified thrombi make the stiff flaps project into the orifice.

In this case the left ventricle is not affected and can pump with ease all the blood it gets; but the left auricle has great difficulty in forcing its blood through this narrow orifice, and therefore it dilates and hypertrophies. The strain falls back on the right ventricle, which now must "push from behind," so to speak; that is, must so increase the pressure in the pulmonary vessels that this will help force the blood through the narrow mitral orifice.

The pulse in mitral-valve disease feels weak, since the wave is small; it is also apt to be irregular in force and in rhythm (Fig. 35). The sounds which the heart makes and the abnormal "murmurs," "thrills," "blows," etc., will lead to diagnosis.

TRICUSPID INSUFFICIENCY.—Tricuspid insufficiency due to disease of the tricuspid valve does occur in children, but rarely in adults. But relative tricuspid insufficiency occurs in practically every case of dilated heart.

The symptoms of tricuspid insufficiency are severe. At each beat the right ventricle forces blood in two directions—through the pulmonary valve, the normal direction, and back through the tricuspid valve into the right auricle. This stream of blood meets the streams from the venæ cavæ and hence impedes the whole venous flow towards the heart. The veins are everywhere overfilled by the dammed-back blood. The patient—especially his lips and fingers—is blue, or "cyanotic." A pulse similar to that sent by the left ventricle through the aorta to the arteries is sent by the right ventricle to the veins. The liver, now swollen to two or three times its normal size by the dammed-back blood, pulsates because of this venous pulse wave. The walls of the stomach and bowels, the kidneys, and all other organs, are turgid with the dammed-back blood, and do not receive their proper supply of fresh blood. Since no internal organ can do its work well, we get symptoms from each. This condition is called "chronic passive congestion" of the stomach, liver, kidneys, etc.—chronic because of its duration, passive because the congestion is the result of blood dammed back into the organ as the result of trouble ahead, and not on account of any trouble in that

organ itself. If the stomach (or kidney, etc.) were inflamed because of local disease, its vessels would be congested, but this would be an acute "active" congestion. The skin of the legs and dependent portions of the body becomes œdematous, or dropsical, or water-logged. Serum collects in the abdominal cavity (ascites) and in the pleural cavities (hydrothorax, or "water on the chest"). If in any way we can make the tricuspid valve competent then circulation will at once improve, the congestion of blood in the various organs will be relieved, and all symptoms due to it will disappear.

PULMONARY-VALVE DISEASE occurs, but is very rare except in children.

Congenital heart disease is sometimes due to an endocarditis which began before birth, but more often to incomplete preparation of the heart for the independent life of the child. Before birth the lungs have no function, and the blood gets its air from the mother's blood. The heart then has several openings not present later,—for instance, one uniting the two ventricles. This should close at birth. When for any reason these orifices do not close, the child has "congenital heart disease." Such a child is a "blue baby,"—blue, or "cyanotic," because the blood cannot get its fresh air. The existence of these children is usually miserable until the circulation has established its normal course.

Myocarditis.—The myocardium is the thick, strong, muscle wall of the heart. In fact the heart is really little more than a myocardium. Myocarditis should mean "inflammation of the myocardium," but real inflammation is rare. It does occur in "blood-poisoning," in which cases even abscesses may form in the heart wall. Again, a very mild inflammation occurs in many acute diseases, such as diphtheria, pneumonia, etc., but "poisoning of the heart" is a better term to apply to the condition produced by the toxins of these fevers, as there is little evidence of a real inflammation of the heart muscle. This is the reason why children who have diphtheria, for instance, are not allowed to sit up or move about vigorously in bed, as they would often like to do; for sudden death is not infrequently the result. The danger-signal in these cases is a rapid, weak pulse, especially an irregular pulse, and a heart which is increasing in size. The treatment for such a case is absolute quiet, while doses of

strychnia, digitalis, etc., will help the heart to recover its strength. In cases of endocarditis there is a real inflammation, which may, and usually does, extend into the myocardium, and cause there a local, genuine myocarditis.

But by myocarditis is usually meant a gradual transformation of the heart wall to scar tissue, and this is the result of arteriosclerosis especially. Such a heart gradually loses the power to contract forcibly; it becomes a weak muscle. It cannot, as in valvular disease, compensate by hypertrophy, for here it is the muscle itself which is at fault. It beats feebly, rapidly, and very irregularly, has little reserve force, and sooner or later some unusual exertion will add the symptoms of a dilated heart. The condition called "fatty heart" is more often a myocarditis than any trouble related to fat.

The Dilated Heart.—The heart is a hollow muscle bag, whose size depends on the "tone" of its muscle fibres. These fibres normally between beats are partially, contracted. This is their "tone." When, because of any disease, the heart is working beyond its limit of strength, the muscle fibres cannot keep up this tone; hence they relax more and more—that is, the heart wall stretches, the heart dilates, and it beats fast, feebly, and irregularly. Soon relative valvular incompetency begins, and the most important form of this is the incompetency of the tricuspid valve. The patient cannot lie down, but sits up, panting for breath; he becomes cyanotic and dropsical; the least exertion is distressing, and his life is one of misery. Yet by proper treatment one may restore the heart to comparatively good health.

In order that heart disease may be treated successfully it is very important that the patient should understand *just what his trouble is*. Perhaps the majority of persons who suppose they have heart disease really have nothing but a neurosis of the heart, which requires different treatment. Strange to say, those with true heart disease have few symptoms which suggest the heart as the diseased organ. If there is a real trouble, the patient should live according to rule; and if he has been warned before a break in compensation, he may live a long, active, useful life, without any trouble from his heart—a life not shortened by the disease. He should live quietly, avoiding any sudden exertion, any strain, or mental excitement; he should exercise as little as possible; he should eat

very sparingly of all foods, especially avoiding large meals, five very small meals a day being better than three ordinary meals; he should keep his bowels open and never allow himself to become constipated; he should avoid all places or conditions in which he might possibly catch cold; he should avoid taking medicine for his heart.

If the patient is in a state of broken compensation, he should invariably have absolute rest in bed, propped up if it is necessary. The drug most used is some form of digitalis, which strengthens the heart muscle. The diet should be as light and as dry as possible, for the heart has to pump around to the kidneys every drop of fluid drunk. A purge (Epsom salts) given every day will keep the bowels in good condition. The water in the chest or abdomen can be removed by tapping, and the venous congestion by bleeding. The nurse is very important later, for after the heart has recovered itself and the patient feels well he wishes to return to active life. Then it is that patience is a virtue; for the longer he remains quiet, so much the better chance has the heart muscle to increase its reserve force, and so much the longer, other things being equal, will the patient be able to stay in active life. Muscular overexertion is especially to be avoided, and very easy exercises may be begun in bed before the patient is allowed even to sit up in a chair.

Compensation restored, the patient should remember that at any time—in a few minutes, even in a few seconds—he can, if he will, ruin the results of his long stay in bed. Foregoing the months or years of comparatively good health that he might have enjoyed, he can by one rash, sudden overexertion, condemn himself to another long stay in bed in as bad, (if not worse) condition as when he was formerly under treatment.

CHAPTER III

DISEASES OF THE RESPIRATORY ORGANS

Internal Respiration.—All the living cells of the body need a constant supply of fresh food, which is sooner or later burned, with the production of ashes. But for combustion oxygen must be present. Among the ashes produced in this process is carbon dioxide, a gas, and this with the other ashes must be removed from the cells as soon as formed, otherwise there is the same danger of the tiny cells' suffocating, that there is of the whole man's dying if his oxygen supply is cut off and the carbon dioxide in the lungs is not allowed to escape. This process of renewing the cells' air, or, more properly speaking, their oxygen, and of removing their "bad air," or their carbon dioxide, is called "internal respiration." It is accomplished by the circulation of the blood. The cells (Fig.

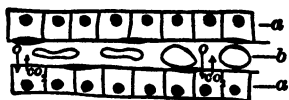


FIG. 37. Internal respiration. *a*, tissue cells; *b*, capillary through which blood is flowing. (The lymph spaces are omitted for sake of clearness. See Fig. 15.) The interchange of oxygen and carbon dioxide is shown by arrows.

37, *a*) are nowhere very far distant from a capillary, *b*, with walls so thin that gas and fluid can freely pass through them. Through these capillaries is constantly passing a stream of red corpuscles whose hæmoglobin is saturated with oxygen. Between the cells and the capillary wall is a thin layer of lymph, (practically the blood-plasma). The tissue-cells take from this lymph all the oxygen it has, and so the capillary is soon surrounded by a layer of lymph which has less oxygen than has the blood in the capillary. The oxygen therefore leaves the hæmoglobin and diffuses into the tissue lymph, where it is at the disposal of the cells. The cells produce carbon dioxide. This diffuses into the tissue lymph, and on into the blood-plasma in the capillary, where formerly there was none. In this way the red arterial blood, rich in oxygen and free from carbon dioxide, changes in the capillary to blue venous blood, rich in carbon dioxide and poor in oxygen.

Venous blood must somewhere have the opportunity to get rid of this carbon dioxide, and to take on a fresh supply of oxygen. This is accomplished in the lungs, the organs of "external respiration."

Reduced to its simplest terms, a lung is an open sac filled with air. In its walls is a network of capillaries so constructed that gas, but not fluid, can pass easily through their walls. Such a lung some amphibians have. It consists of a hollow sac (Fig. 38), and a tube, connecting the interior of the sac with the outer air. The sac consists of a thin wall, on the outer side of which is a thin but close network of capillaries. The wall is of such a nature that oxygen and

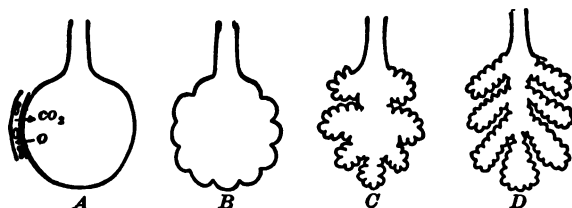


FIG. 38. The simple lungs of amphibians. *A*, the simplest lung, showing a capillary in the wall and the interchange of oxygen and carbon dioxide. *B*, *C*, and *D*, are more complex lungs showing how increase in respiratory surface is gained without any increase in the bulk of the lung as a whole.

carbon dioxide, but no fluid, can easily pass through it. It is a law of gases to diffuse—that is, to distribute themselves uniformly throughout the whole of the space to which they have access. Set free a little bad-smelling gas in one room of a house, and soon it can be smelt in every room. If a large vessel is divided into two compartments by a partition of some porous membrane, and one compartment is filled with one gas, the other with another, then each gas will diffuse through that membrane until each gas exerts the same pressure or "tension" on both sides of the partition. This process of diffusion through a membrane is called "osmosis," and this is constantly going on in the lungs. There is much less oxygen in the blood flowing through the lungs than there is in the air; there is much carbon dioxide in the blood, and none in the air. For this reason these two gases will rapidly diffuse in opposite directions until the blood has almost no carbon dioxide left and has gained much oxygen. There is a chemical attraction between oxygen and hæmoglobin, which

makes them unite to form "oxyhæmoglobin." But this "affinity" is not so strong but that it is easily overpowered in the capillaries by a stronger affinity, which compels the oxygen to osmose into the tissue lymph, where there is an oxygen vacuum. But for some gases this chemical affinity with hæmoglobin is quite strong. When a person inhales in sufficient amounts illuminating gas, for instance, he dies. Illuminating gas contains carbon monoxide, which has a stronger affinity for hæmoglobin than has oxygen. All the hæmoglobin which has become "carbon-monoxide hæmoglobin" is of no further use to the body, and hence if the person breathes enough gas he might as well have lost just so much hæmoglobin, and he will die.

This simple lung (Fig. 38, A) is, theoretically, all that is necessary for a man, but, practically, it would not do, because we have so much blood to "oxygenate." A smooth sac would have to be huge to be big enough to give all the blood, spread out in a thin layer one corpuscle deep, a chance. This difficulty could be met by crinkling and folding up this huge thin-walled sac into small volume, for one can by this method get hundreds of square yards of tissue paper into a small box; but air would with difficulty make its way among all these folds and wrinkles. Other low vertebrates meet this difficulty of furnishing sufficient respiratory surface by lungs shaped as in Fig. 38, B, C, D. A man's lung is really a collection of myriads of small lungs, each of which is separate, and complete in itself. Fig. 39 represents one of our tiny lungs, which consists of a tiny bronchus, *a*, and many "air cells," or "alveoli," *b*. Understand this tiny lung, and you understand our big lungs. Were the respiratory surfaces of all these little lungs united in one big sheet it would cover an area of 90 sq. metres. Were this made into one simple spherical lung it would be a balloon nearly 20 feet in diameter. The tiny bronchi are united like the twigs of a tree to form larger and larger bronchi (Fig. 40) till all of one lung are joined into



FIG. 39. One of the tiny lungs of which our lungs are composed. *a*, terminal bronchus; *b*, air cells.

one bronchus, and these two bronchi of both lungs then unite to form the trachea. For satisfactory respiration one thing more is necessary since this bronchial tree has so many and so fine branches that it would take too much time for the air in the alveoli to be renewed by diffusion from outside. To overcome this difficulty the alveoli expand, sucking in new air; and are compressed, forcing out the old. The lungs are, therefore, put into an air-tight case with distensible walls, the chest. The only opening through which air can

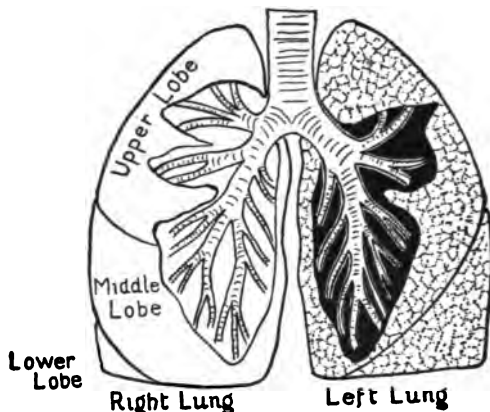


FIG. 40. The bronchial tree. The two lungs are represented as opened, showing the branching of the bronchi. The mottling of the left lung suggests the tiny lungs (Fig. 39) which reach the surface (much exaggerated). The terminal bronchi and fine bronchi are not shown. These fine bronchi unite forming larger and larger tubes until there is but one for each lung, the primary bronchus. These two primary bronchi unite to form the trachea.

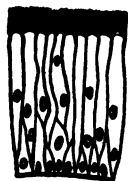
pass into the chest is through the trachea to the interior of the air cells. The chest resembles a pair of bellows. When we distend the chest a vacuum is produced; air must rush in to fill up the vacuum, but its only course is through the trachea and so into the bronchi to the interior of the alveoli distending these. In this way the whole lung is "ventilated"—that is, receives a supply of fresh air which is expelled by the next expiration. The lungs expand passively, following the increased capacity of the cavity they must fill. Respiratory movements are made by the chest wall; their object is to make the cavities of the chest larger and smaller. The lungs do not collapse passively because they are elastic and always on the stretch. If a cut should be made into the chest wall

the person could not breathe. The elastic lung would collapse, and then, as the chest cavity became larger and smaller, air would rush in and out of the pleural cavity, not the interior of the lung, through the hole in the chest wall rather than through the trachea.

Lungs consist of two structures, tubes and sacs. The alveoli (Fig. 39, *b*; Fig. 41), or lungs proper, are little air sacs just about big enough to be seen with the naked eye. They have very thin walls, made up of flat, epithelial cells. On the other side of these is a thick network of capillaries (Fig. 41, *b*). Through this wall gases can easily pass, but not liquids. The sacs' walls are very elastic and this quality allows them to be considerably inflated by each inspiration. The tubes, or bronchi (Fig. 40), have stiff walls, which do not expand and collapse. They are lined by a "ciliated mucous membrane" (Fig. 42). The epithelial cells of this membrane are not flat, as in the alveoli, but "cylindrical," and have their free ends covered by short hairs called cilia, which keep up a constant whipping motion, always in the same direction, and thus sweep out any dust, or excess of the mucus, on this membrane. Many notice that the first thing in the morning they "clear their throat," and expectorate a little gray or black mucus. Truly, this did



FIG. 41. External respiration. *a*, the wall of a sac, consisting of one layer of cells, through which gases can pass, but not liquids; *b*, a capillary. The arrows show the interchange of gases.



Ciliated Surface
Columnar
Epithelium

FIG. 42. Ciliated epithelium (cross section).

“come from their throat;” but this sputum is the accumulated sweepings of the whole bronchial tree, even to the base of the lungs, and this sweeping has been kept up by the cilia all the previous night. Some of that sputum is dust inhaled the day before; some is mucus from the air cells themselves, as well as from the bronchi; and in it are any germs which the cilia may have swept out. It is this sputum that we examine for tubercle bacilli in a very early case of consumption, before the “cough” begins. How hard it is to make our patient believe that there can be any significance in this particle of mucus which he “raises from his throat” every morning.

Diseases of the Upper Air-Passages.—THE NOSE.—The two sides of the nose open externally through the anterior nostrils (Fig. 43, *a*) and posteriorly into the nasopharynx, *b*. These two apertures, front and back, are rather narrow, but between them the nostrils expand into large air chambers completely separated in the centre by the septum (Fig. 44, *a*). The three turbinate bones (Figs. 43 and 44, *c*, *d*, and *e*) project into the nostrils from the sides; the sinuses, or acces-

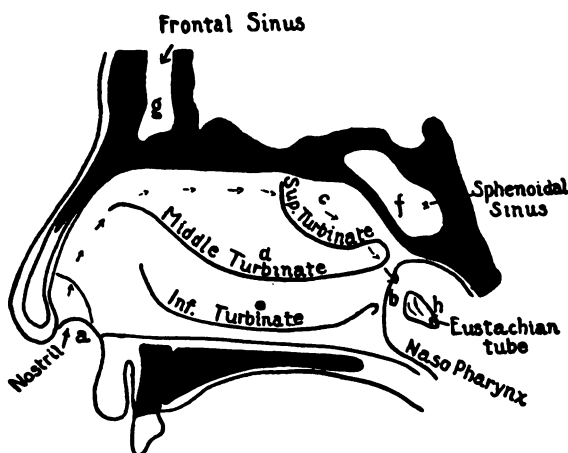


FIG. 43. A vertical section through the nose. Black represents bone. *a*, anterior nostril; *b*, posterior nostril communicating with the nose and the nasopharynx or upper part of throat. *c* superior, *d*, middle, and *e*, inferior turbinate bones. *f*, sphenoidal, and *g*, frontal sinuses. *h*, orifice of the Eustachian tube. The arrows show the direction of the current of air during respiration.

sory air chambers, open on the roof, at the rear and on the sides (Figs. 43 and 44, *f*, *g*, *h*). These sinuses are the hollow interior of the bones of the face. In the lower forehead, above the roof of the nose, are the frontal sinuses (Fig. 43, *g*); along the roof of the nostrils, the ethmoid sinuses (Fig. 44, *i*); opening at the rear, the sphenoid sinuses (Fig. 43, *f*); while the antrum (Fig. 44, *h*), opens on the sides. All of these air chambers, the nose and the sinuses, are a series of communicating air chambers, and all are lined by the same ciliated epithelium.

The turbinated bones (Figs. 44 and 43, *c*, *d*, *e*), which get their name from their shell-like shape, are adapted by shape and position to increase the surface of the air chambers and

to obstruct somewhat the air current. On the superior turbinated bone, *c*, and around it, is the membrane which is the seat of smell.

The air, on entering the anterior nostrils, does not go straight through to the posterior nares, if the former open as horizontally as they should, but passes upward to the roof of the nose, then is deflected downward into the nasopharynx. The current of air is, therefore, considerably obstructed by the turbinated bones and by other irregularities of the nasal wall, and comes into contact with a great deal of mucous membrane. And this is intentional, for this mucous membrane is always quite wet with sticky mucus, and catches practically all the dust and germs in the air that we inhale; its very moist surface moistens the air to the desired amount and thus protects the lungs; it is very full of large blood-vessels which warm the air; its sensitive nerves detect any unpleasant or dangerous odors; its easily irritated surfaces provoke a sneeze or cough to expel any foreign body which comes into contact with



FIG. 44. Cross section through the nostril (black represents bone). *a*, septum of nose; *c*, *d*, *e*, the superior, middle and inferior turbinate bones; *h*, the antrum; *i*, ethmoid sinuses.

it. The air, therefore, when it enters the naso-pharynx, has been rendered suitable for the lungs; it is moist, warm, and almost free from dust and germs. But this is not the only important function of the nose. The air current is considerably obstructed by the tortuous passage of the nose. This is an essential element in the mechanism of respiration, but one not rightly appreciated until we see cases in which it is absent, as in mouth-breathers. (See page 76.)

Bleeding from the nose, or "epistaxis," is common in young "full-blooded" persons. It may also be due to injury, or to the bursting in the nose of blood-vessels which are unusually distended. It is also very often an early symptom of typhoid fever. It is common in conditions with chronic passive congestion.

ACUTE CORYZA, or a "cold in the head," is a catarrhal inflammation of the mucous membrane of the nose. It is sometimes due to irritating fumes, but usually to a bacterial infection of the nose. The germ is not yet known; in fact there are few diseases about which so little is known. The nose seems to be a point of least resistance, with conditions always present for the lighting up of an inflammation. We wet our feet, or a draught of air strikes our back; it is not often in the feet or the back that we suffer, but in the nose. In its mildest grade, and at the beginning and end of a severe case, the mucous membrane becomes congested, turgid with blood, and secretes more of its ordinary mucous secretion than normally. This is soon a clear, thin, glassy, irritating fluid. Then the exudate begins; pus-cells and perhaps also a few red corpuscles pour out from the capillaries, and the secretion becomes yellow. The swollen mucous membrane fills up the air-passages; for there is normally but little free space in the nose, and a very slight swelling can entirely shut off nasal respiration. The orifices of the tear-ducts are in the nose and are closed by the swollen membrane, and so the "eyes run." The mucous membrane is very sensitive now, and hence we sneeze often to keep the nose clear from the exudate. This trouble is due to an infection, and hence there are fever, headache, etc., for a day or so. The exudation and swelling of the membrane gradually subside during three or four days, unless the trouble extends to the throat and lungs.

One should remember that an acute coryza is often the first stage of measles, of whooping-cough, and of influenza, and should watch for the signs of these troubles in a child who has been exposed to them.

For treatment the patient is kept quiet until the acute stage is over. A calomel purge is given at once. Some local remedies relieve the trouble, especially local sprays,—adrenalin, *e.g.*, which lessens the swelling in the nose.

Hay fever is an especial variety of acute coryza. It is more severe than a simple cold in the head, there is often bronchitis or true asthma with it, and the general symptoms—headache, malaise, mental depression, etc.,—are more pronounced. The cause of this trouble is a nasal mucous membrane more sensitive than normal to certain irritating substances, as the pollen of plants (especially those of the rye

family). These pollen grains contain a very irritating poison to which these persons are especially susceptible.

Some persons have attacks in the spring ("June cold," "rose cold"), others in the fall ("autumnal fever"), and in regions without winter—for the first frost cuts an attack short—the hay fever may last all the year.

Fortunately for the average hay-fever case, who hates to be called nervous, it is now agreed that the nervous form of hay fever, the form whose attacks are brought on by the smell of a certain flower, or animal, etc., is a different trouble.

There are some persons who complain bitterly because their noses frequently stop up. This may occur suddenly at night or in the day, sometimes without warning, sometimes following some odor, a breath of cold air, or some mental or nervous stimulus. This is usually due to a local nasal trouble, a spur or polyp in the nose, but sometimes is a signal of trouble further away.

The treatment of hay fever is to stay, during the season when the hay fever attacks a person, in some region where he is free from attacks. For most persons this is the dry mountain air. The hypersensitive nasal membrane can be destroyed, but the result is sometimes worse than the hay fever. But the true hay-fever patient must remember that, although his attacks themselves are not neurotic, yet the hypersensitiveness of the nasal mucous membrane is a first cousin to a neurosis, and he must not take offence if a general building up of his nervous system is suggested.

CHRONIC CORYZA.—Many persons suffer from "chronic cold in the head," or chronic catarrh. There is always an abnormally large amount of connective tissue in the mucous membrane of their noses. In acute coryza the membrane may swell excessively, but this swelling is due to an excess of blood and a water-logging of the membrane with lymph. All this can quickly disappear and the membrane return to its normal thickness. In the chronic form the membrane becomes permanently thickened by new tissue and even by a thickening of the bone. The result when local is a "nasal spur" on the septum, or a "hypertrophied turbinate" when these bones are affected.

When for any reason the nasal secretion can accumulate in the nose, local hypertrophies are rapid in their formation.

When the nasal infection creeps into one of the various sinuses and sets up a chronic inflammation with pus formation, the condition is more serious; for in this sinus the infection may be active for months or years. If the trouble is in the frontal sinuses, there is a continuous discharge from the nose; if in the sphenoidal sinus, pus will trickle into the throat. The constant irritation of the pus makes the mucous membrane grow thick, or hypertrophy, and also leads to polyp

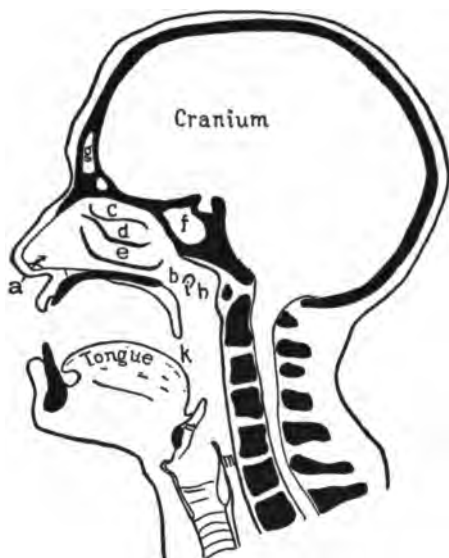


FIG. 45. Vertical section through the skull. *a*, anterior nares; *b*, posterior nares; *c*, *d*, *e*, superior, middle and inferior turbinated bones; *f*, sphenoid sinus; *g*, frontal sinus; *h*, nasopharynx; *i*, Eustachian tube; *k*, position of tonsils; *m*, esophagus; *n*, epiglottis over larynx.

formation, to the growth of nasal spurs, etc. The treatment is to open and drain the sinus, and this may mean an operation.

Another chronic infection of the nose leads to wasting, or atrophy, not alone of the mucous membranes, but also of the bones themselves. The result is that the nostrils are large empty caverns. The exudate, which sticks to the walls in large quantities, has an exceedingly repugnant odor, which can be detected at a distance, and which makes conversation with the patient quite disagreeable. Fortunately for the patient, he smells nothing. This condition is called "ozæna."

ADENOID GROWTHS.—The *nasopharynx* (Figs. 43 and 45, *h*) is the upper part of the pharynx and is just behind the nose. Into it open the posterior nostrils, *b*, and the Eustachian tubes, *i*, from the middle ears. The most important and commonest trouble which occurs here is the growth of adenoids.

By “tonsils” we usually mean two almond-shaped bodies in the back of the mouth, at *K*, Fig. 45, but these, although the largest and most easily seen, are only part of a circle of

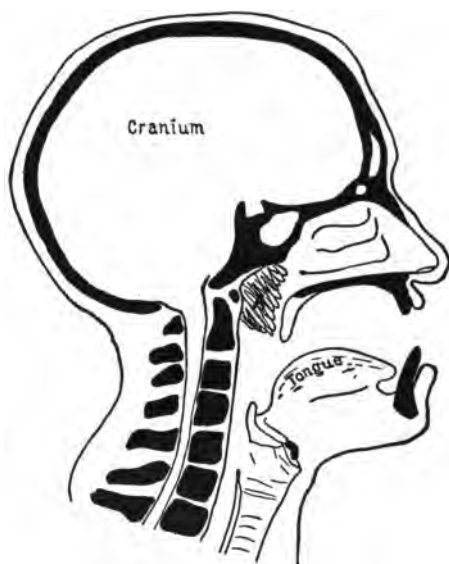


FIG. 46. Vertical section through the skull showing a mass of adenoids, *A*, in the nasopharynx.

tonsils which crosses the root of the tongue and the roof of the nasopharynx, thus completely encircling the throat. The pharyngeal tonsil, at the roof of the nasopharynx, is always present in children, but soon disappears. If it becomes unusually large it may fill up the nasopharynx completely (Fig. 46, *A*), and be visible hanging down behind the soft palate (Fig. 46). This tonsil is just as dangerous a portal entry for infection as are the throat tonsils. That phase of the subject we discuss under the heading “Acute Tonsillitis” (page 300). We now refer to the trouble adenoids, filling the

posterior nares, can cause by virtue of their presence. They cause a surprisingly long list of troubles. They obstruct the nasal respiration at an age when the bones of the face and chest are growing, are soft, and hence are easily influenced. The base of the nose becomes broad and prominent and the nostrils narrow. The eyes are far apart. The expression is rather stupid. The hard palate of the mouth becomes high-arched; the upper jaw becomes narrow; the upper front teeth project "rodent fashion;" the voice is nasal. The sternum becomes prominent—"pigeon breast;" the lower margin of the chest is deepened ("Harrison's groove"). It is strange that parents should not realize that the difference between some very pretty and some homely faces, between some well-shaped and other deformed chests, is due to adenoids, and that the child's beauty could have been preserved. The adenoids also block the orifices of the Eustachian tubes. The result is often chronic otitis media with slowly developing deafness, and sometimes acute suppuration of the middle ear—"running ears"—with the blood-poisoning, the abscesses, the bone and joint inflammations, which may follow.

The pharynx, the larynx, and the lungs suffer from adenoids, because the air is not so well warmed, and saturated with moisture, not so thoroughly freed from dust and germs, by the mouth as by the nose, and chronic laryngitis and chronic pharyngitis are the logical result. The child does not sleep well, but has bad dreams, wakes suddenly with a "night cry," and is found sitting upright in bed. A more important result is asthma, and this serious trouble may continue throughout life, even after the adenoids have been removed or have disappeared. The result of asthma is emphysema of the lungs, a serious condition. Lastly, and for some reason which we don't know, children with adenoids do not develop mentally so well as other children. They not only look stupid, since they are mouth-breathers, but they sometimes really are stupid. These adenoids can be easily removed by a simple and satisfactory operation, and if it is done in time all these results can be prevented.

LARYNGITIS.—The nose and nasopharynx are lined with the same kind of ciliated epithelium as the larynx and whole bronchial tree, but the pharynx, or throat, the place where the respiratory and alimentary passages cross each other (Fig. 45, *m*,

n, and Fig. 47), is lined with "flat," "squamous" epithelium. This is fortunate, for the tendency of inflammation of epithelial surfaces is to spread. A bronchitis of the finest tubes will quite certainly spread up to the larynx; a laryngitis will tend to spread down to the bronchi. But this strip of epithelium of a different character acts as a barrier, and an acute coryza does not always spread to the throat, an acute bronchitis seldom spreads to the nose. An acute inflammation of this ciliated epithelial membrane is similar to that described as coryza, no matter what part of the respiratory

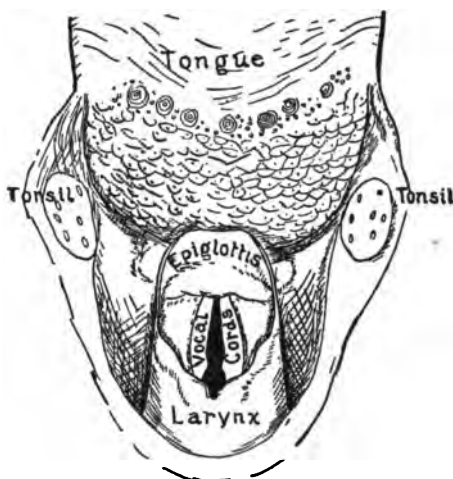


FIG. 47. The throat (viewed from above and a little behind).

tract is affected. We have, first, hypersecretion of a clear mucus, then the mixture of pus and red cells, then the profuse discharge of watery pus, then the gradual diminution of the leucocytes till the abundant but still diminishing exudate is pure mucus, then the disappearance of this also. The membrane is first moist, then red; the epithelial cells are often cast off in large sheets, leaving the bleeding suppurating submucosa bare; then this gradually becomes re-covered with epithelial cells. When this process affects the larynx, we have "acute laryngitis;" when the trachea, "acute tracheitis;" when the bronchial tree, "acute bronchitis." But there are differences in symptoms; these depend on the

location of the process. In *acute laryngitis* speech is difficult, and the cough is especially severe. It is relieved by resting the voice, by breathing air moistened by steam, by an ice-bag on the throat, and by local soothing sprays. *Chronic laryngitis* is due to repeated attacks of the acute trouble, caused by frequent inhalation of irritating gases, as tobacco, and by habitual overuse of the voice, as by the street huckster.

ŒDEMA OF THE LARYNX is a very serious condition, for the patient may suffocate. The larynx is a stiff box of cartilage and will not stretch. The orifice, called the "glottis" (whence the synonymous term "œdema of the glottis"), through which the air must pass, is really quite narrow, and a little swelling of the mucous membrane may close the orifice tightly. This occurs rarely in acute laryngitis, less rarely in severe inflammation of the throat, as diphtheria, erysipelas, scarlet fever, and often when there is an ulcer there, as in tuberculosis, typhoid fever, etc. It also is often the cause of death in Bright's disease. In some patients with "giant œdema" this may be the point which swells. The patient suddenly becomes more and more dyspnoëic; he struggles to draw each breath. The voice is at first husky, then lost. Unless a spray of adrenalin is applied, or the throat is opened below the larynx, or an ice-bag is put over the throat, the man may die in an hour or so. If the operation of tracheotomy had then been known, George Washington's life might have been prolonged.

An attack somewhat similar, but of very different nature, occurs in neurotic children under three years of age, especially in babies with rickets. It is due to spasms of the muscles of the throat. The child struggles for breath; the face becomes blue; then all of a sudden the spasm is relieved, and the child takes a deep breath with a crowing sound. This condition is called **LARYNGISMUS STRIDULUS**, or, in popular terms, "child crowing" or "passion fits." It is a nervous trouble. The general condition of the child should be built up.

"**SPASMODIC CROUP**" is thought to be a similar condition. The child awakes at night with sudden difficulty in breathing, a croupy cough, husky voice, and congested face. In a short time the breathing is suddenly relieved. This may recur for several nights. During the day there will be a mild bronchitis. Various remedies will relieve the spasm—a warm bath, an

emetic, a whiff of chloroform, etc. The larynx is often attacked by tuberculosis, and this condition is very serious. These patients are hoarse, have the worst cough of all, and suffer terribly when they try to swallow. Cancers of the larynx are common.

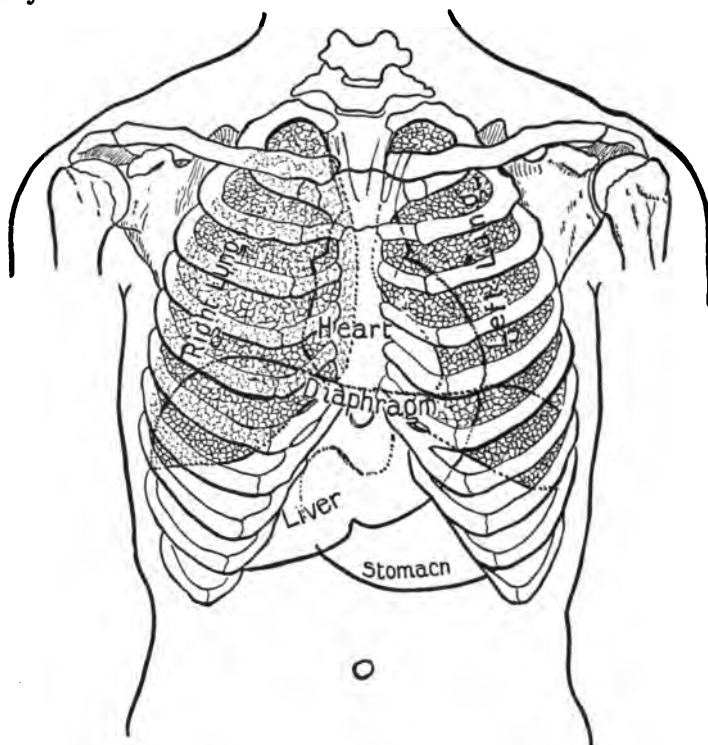


FIG. 48. A diagram of the normal thoracic and upper abdominal organs.

ACUTE BRONCHITIS, or a "cold in the chest," is an acute inflammation of the mucous membrane of the bronchial tree. It may extend down from the nose or from the larynx, or the person may catch a cold "on the chest." It may be an early symptom of more severe diseases, such as measles, whooping-cough, typhoid fever, or influenza. This inflammation may, especially in children, extend along into the air cells, and set up a broncho-pneumonia. The symptoms of acute bronchitis are pretty general—that is, fever, headache,

malaise, languor, various general pains; and local, such as a cough, painful and dry at first, then easier and loose, with abundant sputum. The course of a simple case is about seven days. The treatment, apart from the various medicines which may be prescribed, is rest in bed till the fever is all gone, warm baths, and a mustard plaster on the chest. Often an ordinary kettle boiling in the room will keep the air moist and make the patient more comfortable.

CHRONIC BRONCHITIS is one of the most common and troublesome of maladies, especially for the aged. The mucous membrane of the bronchi has, as the result of long-standing trouble, become thicker or thinner than normal, and often is very sensitive. Such patients have a "chronic cough"—some a "winter cough," some a "morning cough." Some raise almost no sputum; some, vast amounts of a watery sputum; some, a very fetid sputum ("putrid bronchitis"); some expectorate only in the morning and "clear their tubes for the day." But chronic bronchitis may be a complication, or a symptom of a more serious trouble, such as chronic lung disease (emphysema, *e.g.*), or heart disease or Bright's disease or gout, etc.

These patients should choose that climate in which they have least trouble; should so dress as to avoid catching fresh colds; and must be sure that there is behind their bronchitis or "catarrh" no other trouble which needs treatment. Among drugs, potassium iodide is especially valuable to relieve the dry cough.

FIBRINOUS BRONCHITIS is a remarkable condition, in which the bronchial walls excrete fibrine. This patient will expectorate tree-like casts of the bronchi.

ASTHMA to the popular mind means recurring attacks of shortness of breath. But such may also be due to diseases of the heart ("cardiac asthma") or of the blood-vessels and to Bright's disease ("renal asthma"). In true asthma the trouble is in the finest bronchi. These, either because of swelling of the mucous membrane (and these little tubes are so fine that a slight swelling will almost close them), or because of spasm of the muscle fibre in their walls, may become so narrow that air can scarcely pass through them to the air-cells, or, to speak more properly, can scarcely be squeezed out of the air-cells. In these cases, although each

inspiration is very difficult, each expiration is still more so. The patient tries to squeeze the air out of his lungs by forcibly contracting the muscles of expiration, and makes a loud wheezing sound when doing this. Asthma surely is partly a nervous disorder, and hence is called "hay fever of the lungs;" but there is often bronchitis with it—that is, an inflammation—so that we are sure it is not entirely a neurosis.

In many cases asthma has a "reflex" origin; that is, the real disease is somewhere else, but through the nervous system produces its symptoms here. For instance, a person often has asthma of the lungs because he has adenoids behind his nose. Or he has asthma because he has a polyp in his nose. Remove the adenoids or the polyp, and then the asthma will disappear. The asthmatic attacks are often "touched off"—for they resemble a convulsion in many ways—by some trouble in the stomach or, especially, in the pelvis. Certain odors, a cold breath, dust, etc., may bring on an attack.

Asthmatic attacks come on suddenly, usually at night. The patient feels chilly, has a tight feeling in the chest, has difficulty in breathing. He sits up in a chair, looks anxious, and begins to perspire freely. He takes slow, deep breaths, struggles to draw the air into the lungs, works harder to blow it out. The respirations are loud and wheezing.

This lasts a few minutes or hours, and then comes relief; but there will still be some wheezing for a few hours or days, and a bronchitis will develop, with cough, and an expectoration in which can be found the prettiest strings of twisted mucus that one ever sees. These attacks may come on night after night or on very rare occasions. The results of these attacks are serious, for the lungs become emphysematous from the chronic bronchitis, and even the shape of the chest changes.

The cause of the attacks must be found and removed. This means that the nose, the throat, the pelvis, and the genital organs must be carefully examined and any trouble there corrected. The patient soon learns not to eat heartily, or late at night, and to avoid cold draughts, and any odors, etc., which precipitate the attack.

He usually keeps himself armed with "perles" of amyl nitrite, or a small bottle of chloroform, or paper steeped in potassium chlorate, belladonna, or henbane, the fumes of

which, when burned, will relieve the attack. Between attacks potassium iodide will aid much.

Diseases of the Lungs.—**EMPHYSEMA.**—This condition is easily understood if one remembers that our lungs are a myriad of tiny lungs, each about $\frac{1}{16}$ inch in diameter, with elastic walls, which are inflated to larger size by each inspiration, and collapse because of their elasticity during each expiration. Suppose now that the walls are naturally not

very elastic, or are abnormally weak, or are repeatedly over-distended. Then they will lose their elasticity more and more, and will remain inflated; their weak walls will become weaker until some unusual respiratory movement will burst them, and two or more air cells will then become one large one. Thus the process continues. The lung becomes less elastic and more and more inflated; more and more of the air cells break down; the air cells become larger until some are 2 or 3 mm. in diameter; and in places the lung contains cavities even as large as a hen's egg. Fig. 49, shows below, air-cells of normal

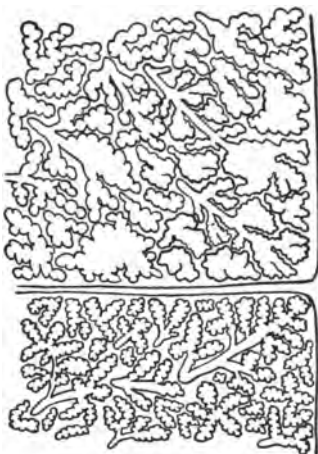


FIG. 49. Emphysema of the lung. Above is a portion of a very emphysematous lobe of the lung. Below is represented for comparison a portion of a normal lobe. Compare with Fig. 39.

lung cut across; and above, those distended as in emphysema.

In emphysema each breath requires an unusual amount of muscular force, for the patient must force the air out of the lungs. Inspiration is short and quick, expiration long and wheezing. He will, therefore, be "short of breath" and have asthmatic attacks. The chest will become thick and barrel-shaped. Another result is that there is much less respiratory surface because so many walls of alveoli have disappeared, and, of course, their capillary network also. These patients always look blue, or "cyanotic." It requires an unusual amount of work for the heart to pump the blood through such lungs, and hence the right side of the heart hypertrophies. And, finally, there is usually considerable bronchitis.

Emphysema of the lungs occurs in men who do a great deal of hard blowing, as horn players, glass blowers, and in men whose daily work is hard muscular labor. But in these men the emphysema is seldom of as severe a grade as that occurring in children with adenoids or in adults with asthma.

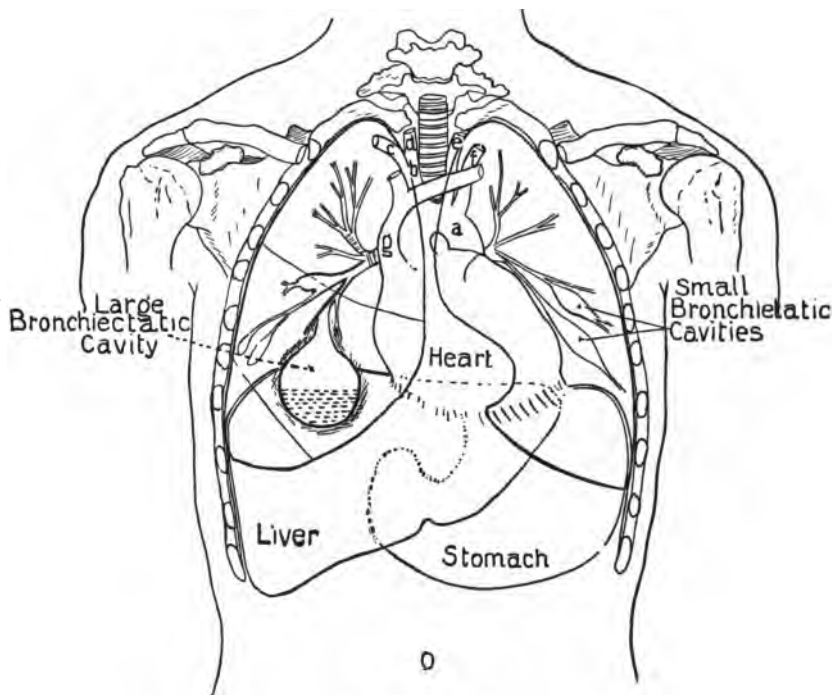


FIG. 50. Lungs the seat of bronchiectasis. *a*, aorta; *b*, innominate artery; *c*, right subclavian artery; *d*, right common carotid artery; *e*, left common carotid artery; *f*, left subclavian artery; *g*, superior vena cava. In this figure the pericardium is represented as not opened.

The worst cases may be best explained by the inheritance of poor lung tissue.

For emphysema, as for asthma, the best treatment is to remove the cause—the adenoids, etc. These patients will from necessity lead a quiet life. The chronic bronchitis especially needs treatment.

BRONCHIECTASIS (Fig. 50).—This condition is usually the result of chronic bronchitis. The bronchi are tubes with walls

made rigid and strong by rings of cartilage. When, because of disease, these walls become weak, the lumen of the tube becomes dilated, and a sac-like cavity develops. These cavities differ from those of tuberculosis, for their walls are those of the bronchi. These cavities are distended bronchi, while in tuberculosis there are death and decay of lung tissue, the expectoration of which leaves a hole behind. Sometimes one or a few bronchi become much dilated, a condition known as "saccular bronchiectasis." If a great many bronchi become just a little dilated the condition is called "diffuse," or "cylindrical" bronchiectasis. The weakening of the wall may be due to a very severe, long-standing bronchitis which erodes the mucosa and cartilages. Many cases date back to an attack of grip. But not only must the bronchial wall become weak, but there must be a force to inflate the bronchial cavity. Repeated coughing can do this; the accumulation of exudate in the growing cavity can, by its weight, do this; and the bands of scar tissue, which in pulmonary tuberculosis radiate out from the bronchi, can, by contracting, pull out these walls. The symptoms are those of chronic bronchitis unless one or more cavities are very large.

These large cavities may hold a cupful or even more than a pint. The sputum collects in them, and then, on some change of posture—usually on rising in the morning—a little sputum flows out into a normal bronchus. Then the patient coughs until he has emptied this sac. The expectoration of large amounts of sputum, in paroxysms, usually means saccular bronchiectasis. These patients often have hemorrhages from these cavities, sometimes very severe, and the odor of the sputum is often very foul. If there are many small cavities there may be no symptoms to distinguish this condition from chronic or putrid bronchitis.

The treatment is that of bronchitis. The odor of the sputum can be much reduced by creasote inhalations. Out of a sheet is made a tent, under which is a chair on which the patient sits. Under the chair is a small lamp which warms a basin of water containing one teaspoonful of creasote. These fumes inhaled for twenty minutes or an hour every day relieve the cough and make the odor of the sputum much less objectionable.

CHAPTER IV

DISEASES OF THE UPPER ALIMENTARY TRACT

DISEASES OF THE ŒSOPHAGUS

THE ŒSOPHAGUS (Fig. 45) is a muscular canal about nine inches long, through which the food passes from the pharynx to the stomach.

Sometimes the œsophagus contracts strongly, and for a time food cannot pass through it. This occurs most commonly in nervous persons, but the best-known illustration is hydrophobia. In other cases there is a real stricture of the œsophagus. In over eight-tenths of the cases this is a cancer. These patients really starve to death unless an artificial opening is made into the stomach, through which the patient can be fed. Another common cause of the constriction is the scar following the ingestion of lye, or of previous ulcers. In these cases the œsophagus may be greatly dilated above the stricture, and the food eaten may be vomited. When the vomitus is not acid to litmus, this condition may be suspected.

DISEASES OF THE STOMACH

The gastro-intestinal canal (Fig. 51) is a tube about twenty-five feet in length, through which the food passes, and in which it is acted upon by various digestive fluids. By "digestion" we mean "liquefaction." The food which is in the stomach or bowel is not *in* the body; it is enclosed by the body, but is really outside it. A coin held in the mouth, for instance, could not be said to be any more within the body than a coin held in the closed fist is inside the hand. To get inside the body the food must, first, be rendered liquid, that is, soluble in water. After this it is absorbed through the walls of the intestine, enters the blood-vessels and lymphatics, and then is truly within the body. That part of the food which cannot be absorbed is the fæces. Nearly all of the food we take is solid and insoluble. The process by which this is rendered liquid and soluble is digestion.

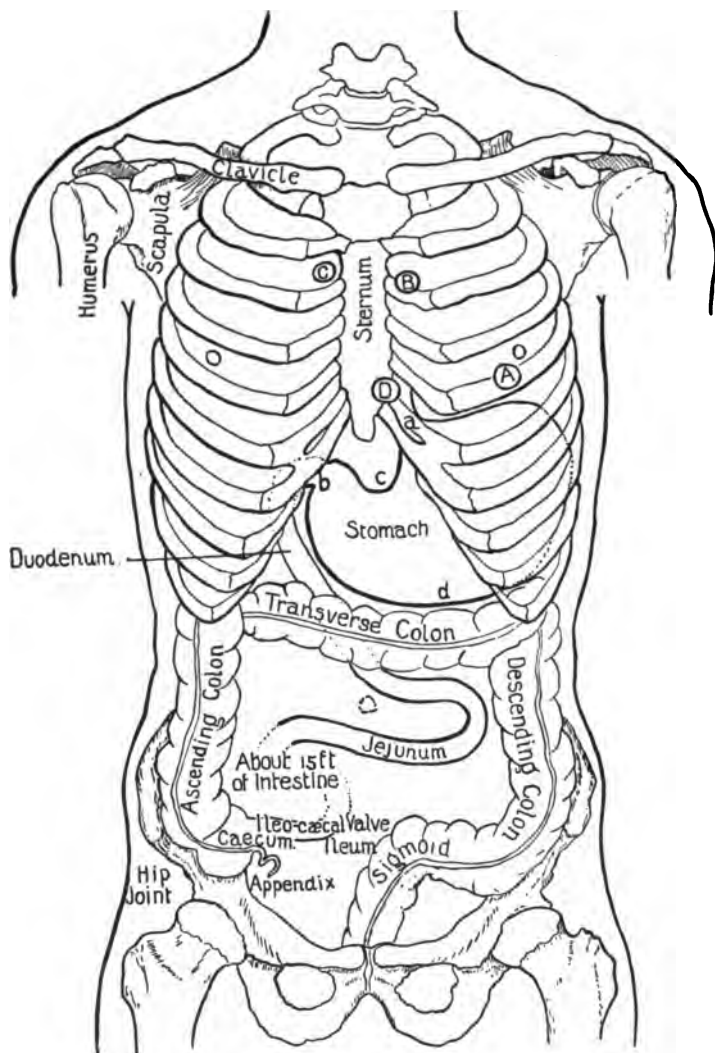


FIG. 51. A diagram showing the position of certain organs. *A*, the point of maximum impulse of the heart, also called the "mitral area." *B*, the "pulmonic area." *C*, the "aortic area," and *D*, the "tricuspid area." The valves are not situated under these areas, but sounds made by these valves are best heard there. *a*, cardiac orifice; *b*, pylorus; *c*, lesser, and *d*, greater curvature of the stomach.

The food-stuffs consist of water, which is unchanged in the gastro-intestinal canal, and is absorbed and excreted as such; of inorganic salts, which are also absorbed practically unchanged; of carbohydrates, fats and proteins. The carbohydrates are for the most part soluble in water, but are not in a form useful to the body. The only kind of sugar which our body uses is glucose, or the sugar of grapes, which occurs only sparingly in our food. The number of carbohydrates (sugars and starches) which we eat is great. In the process of digestion these complex sugars are broken down to the simple sugars, such as dextrose, levulose, maltose, etc., and then these are "inverted"—that is, are changed over into glucose by a change in the internal arrangement of the molecule. The fats are insoluble in water. They are composed of glycerin and fatty acids. Digestion splits this combination. The glycerin is easily absorbed, and the fatty acids unite with alkalies to form true soaps, which also are easily absorbed. Proteins and albuminoids are very complex substances, and in the forms eaten are not soluble in water. By digestion they are split into many very much simpler substances, such as peptones, amido-acids, etc., which are soluble in water and so are absorbed by the intestinal wall.

Once within the intestinal wall, all of these simple substances are reconstructed. The glycerin and soap are again united into a fat. All of the simple products of protein digestion are united together to form new proteins—not the same as before, but those adapted to the uses of the body. It is very much the same as though one were to tear down a house and with all the bricks, timbers, and other building material, were to reconstruct another house of different architecture. We eat a multitude of various proteins. The bricks, timbers, etc., of which the various proteins of beef, pork, lamb, chickens, ducks, vegetables, etc., are composed are much the same in all. They differ in relative amounts and in arrangement, just as the difference in the house depends on the amount and arrangement of its building materials. In the intestinal wall all these various building materials are rearranged into the few proteins of which our body is composed.

By food we mean substances necessary to the normal composition and functioning of the body. Withdraw water, salts, etc., and the body will die. Some foods—and this is

true of the sugars and fat—are used as coal in a furnace, that is, to produce heat. They may remain for years stored up in the body like coal in bins but they are never more than fuel. Other foods are used to replenish the worn tissues of the body, just as an engine needs new steel parts when it is worn. The steel becomes a part of the engine, the coal never. So the proteins which we eat may become a part of the body, that is, of the living protoplasm; the sugars and fats, never. Various foods are used as raw material out of which the body can make new substances. For instance, the gastric juice does not exist in the body as such. It is manufactured by the stomach from the various articles of food. Sooner or later all food, except water and salts, is burned, and ashes are produced, just as ashes result from the burning of coal in the furnace. But this process is best described in another chapter.

In the mouth the food receives its first preparation for digestion. It is ground up into small fragments and mixed with saliva. The ptyalin of the saliva starts the starch digestion. The food is in the mouth for a few seconds only, but the salivary digestion continues in the stomach normally for about thirty minutes, or until enough acid is secreted to destroy the ptyalin; and the result is that about one-half the carbohydrate is transformed into simple sugar.

There is much truth in the old German proverb that “well chewed is half digested,” for by a few minutes spent in masticating the food we spare the digestive organs a great amount of work. Gladstone considered that his good health was largely due to the fact that every mouthful of food was chewed 32 times, once for each tooth. Recently long chewing has become quite a fad, and in honor of the man who has written a good deal about it this is called fletcherizing. The importance of the teeth in the maintenance of good health is well illustrated by the improved health which old persons now enjoy, as compared with the health of those who were aged formerly, when false teeth were neither so well made nor so easily obtained as at present. Another reason why thorough mastication is desirable is that it stimulates the secretion of gastric juice.

Fluids pass quickly through the cesophagus and at once enter the stomach, but masses of solid food are held, by the

muscle ring, just above the stomach for about six seconds. This ring then relaxes and allows the food to drop into the stomach. This is one reason why a person should eat slowly and not swallow oftener than once in about six seconds, for we know that if one bolus of food is waiting to enter the stomach, and another is then swallowed, the first bolus does not drop into the stomach at the end of its six seconds, but waits through the six seconds of the second

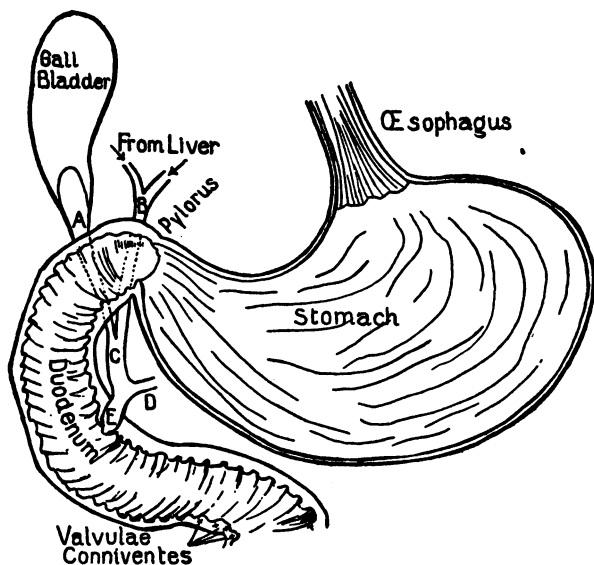


FIG. 52. The normal stomach, duodenum, and gall ducts. A, the beginning of the cystic duct. B, the hepatic duct. C, the common duct. D, the pancreatic duct.

bolus, etc. Hence, in rapid eating the oesophagus can get overfilled.

The stomach (Fig. 51) is a large, hollow receptacle, which holds normally about 1600 c.c. It lies in the upper abdomen, almost entirely on the left side of the body, and for the most part tucked away beneath the ribs. It does not at all occupy the position that popular opinion holds when a man says "he has a pain in his stomach." It has two orifices: the cardiac orifice, *a*, where the oesophagus enters, which lies from about 2 to 3 cm. to the left of the mid line and deep in the abdomen behind the 6th or 7th costal cartilage, and is

provided with a circular muscle which can close it completely; and the pylorus, *b*, the communication between the stomach and the bowel, which lies more superficially—sometimes in the mid line just below the ensiform cartilage, and sometimes 2 or 3 cm. to the right of this. The lesser curvature, *c*, or upper margin of the stomach, is from 3 to 6 inches long. The stomach at its widest point measures from 4 to 5 inches. The lower margin of the stomach, *d*, is called the “greater curvature.” This crosses the mid line about one-half way between the ensiform cartilage and the navel.

GASTRIC JUICE.—The first food of a meal enters an empty stomach. Very soon the walls of the stomach begin to secrete gastric juice, and at the same time the churning movement, peristalsis, begins. This mixes the food, churning it back and forth from one end of the stomach to the other. The gastric juice contains pepsin, a ferment which splits protein, rendering it soluble; hydrochloric acid, which is necessary for peptic digestion; and rennet, which clots milk. Gastric juice is no constant fluid, but differs according to the meal to be digested.

The peristalsis, or churning motion of the stomach, keeps the food, now called chyme, in constant motion. It constantly brings the food into contact with fresh gastric juice. As the mass of food is pressed towards the pylorus, the portion which has already become fluid is allowed to enter the intestine, but that which is still solid is retained in the stomach until it is fluid. If at the end of a few hours any food is still solid, it is allowed to enter the intestine. Gastric digestion takes from one to seven hours. If at the end of seven hours after a meal there is anything left in the stomach, something is wrong. During these few hours the protein is about half digested, a little of the fat has been split, and about half of the starches is digested.

In the stomach practically no food is absorbed; all this is passed on into the bowel, for it is not the function of the stomach to absorb digested food. Alcohol, however, and certain drugs can be absorbed through the stomach wall.

Gastric juice is not absolutely necessary. The stomach may secrete none and yet the patient have no symptoms. Also, a man can live even without a stomach. The question arises, therefore, what is the stomach for? In the first place

it is a reservoir in which is stored up the food eaten occasionally and in large amounts. This is here prepared for the intestine and allowed to pass on into it in very small quantities at a time. Thus the bowel is supplied with food in just the amounts which it can handle. Also, the germs in the food are practically all killed by the acid gastric juice. And, finally, in the stomach about half of the solid matter is digested—that is, changed to a liquid. Remove a man's stomach, and he would have to eat only very soft food and in very small amounts, and hence very frequently. He would spend the most of his time eating. With a stomach he can eat occasionally large amounts. Gastric juice is not secreted simply because food lies in the stomach. Solid food might remain there for hours and not a drop of gastric juice be furnished to digest it. Some is secreted in response to a nervous stimulus. For instance, the odor, appearance, or taste of appetizing food stimulates, through the nervous system, the secretion of gastric juice. It is for this reason that we should enjoy the food we eat, and should chew it longer than we usually do. When we do not enjoy our food—either its appearance, or its odor, or its taste—there is not nearly so much gastric juice secreted as might be the case, although the food may be perfect in its nutritional qualities. This psychic stimulus produces what is known as a "primary secretion" of gastric juice. If some of the food is in liquid form, this soaks into the wall of the stomach and there chemically stimulates the secretion of gastric juice. This is one of the chief values of soups, gravy, and sauces. This is called "secondary secretion," and is caused also by the soluble products of the digestion, which the gastric juice of the primary secretion furnishes. The secondary secretion is really the more important of the two. The secretion of gastric juice, and, therefore, digestion, depend much on the mind of the eater. A person who enjoys his food secretes more juice than a person who does not. If one is worried or anxious or frightened, there will be no nervous stimulus of secretion. The discovery of the importance of these two stimuli, the mental and the chemical, we owe, you will read in books, to a Russian by the name of Pawlow. But, as a matter of fact, this discovery was made centuries ago by some intelligent boarding-house keeper, who served soup as the first course. Suppose that at her table sat four men. Mr. A.

enjoyed his food hugely, and so had a good primary secretion. Also, he ate all the courses, including the soup, and so had plenty of chemical stimulus to secretion. Mr. B. enjoyed the solid food, chewed it well, but did not take soup. He probably had no indigestion, because the mental enjoyment of chewing the food furnished considerable gastric juice, and this soon digested enough of the solid food to cause also the chemical stimulus. Mr. C. did not enjoy his food at all, but did always take the soup. Although there was no mental stimulus to secretion, the soup was sufficient chemical stimulus; and so he soon had enough gastric juice, and therefore escaped dyspepsia. Mr. D., however, did not like his food and did not take soup. There was no mental stimulus to secretion, and, since he ate only the solid food, there was no chemical stimulus. His food rested undigested in his stomach, and he was sooner or later a confirmed dyspeptic.

ACUTE GASTRITIS.—Acute gastritis is a complaint so common that we need not take much space to describe the symptoms. It is often due to some error in diet. The child eats too much or too rapidly, or eats food which is unsuitable because easily fermented or partly decomposed. The stomach cannot be expected to stand this abuse, and so becomes somewhat inflamed. But acute gastritis also is often the first sign of an acute fever, *e.g.*, typhoid fever. The best example of acute gastritis, however, is that due to alcohol.

The gastric mucous membrane of such a stomach is red and swollen, it secretes little gastric juice, and this contains very little acid but much mucus. The patient has uncomfortable feelings in his abdomen, with headache, lassitude, some nausea, often vomiting. This vomiting relieves him considerably, for it removes the irritating substance. The tongue is coated, and the flow of saliva is increased. If this decomposing, fermenting, irritating mass is not vomited, but reaches the bowel, colic and diarrhoea are the result. As a rule the patient is well in about one day, although he may not have very much appetite for the next two or three days.

The treatment is to get rid of this irritating food as soon as possible. If it is still in the stomach the patient can drink warm water, or mustard and water, or stick his finger down his throat, and so induce vomiting. If it is in the bowels, one gives the patient castor oil or calomel in large doses, in

order to empty them as soon as possible, and this is wise even if the patient has diarrhoea.

CHRONIC GASTRITIS.—Chronic gastritis is a diagnosis often made, but usually incorrectly. Most of the cases thus diagnosed are really cases of nervous dyspepsia. In true chronic gastritis the wall of the stomach is really diseased. Its mucous membrane is a little thinned, sometimes much—so much that there is scarcely any mucosa left; and the muscle wall is a little weakened and hence less able to empty the stomach, which, as a result, becomes dilated. It secretes too much mucus, and a gastric juice abnormal both in quantity and in quality.

Among the causes of chronic gastritis, foolish habits of eating are especially important. The stomach is a long-suffering organ, and it is remarkable that it stands as much abuse as it does. Many persons overeat. Others eat food which has not been sufficiently masticated. Some eat large amounts of indigestible foods; others eat food good in quality, but improperly prepared, and in this connection we may say that the frying pan is perhaps the commonest cause of chronic gastritis, for food that is fried is very hard to digest. Some indulge for a long time in excessive amounts of fats or carbohydrates. Others overeat improper food, and in this connection should be mentioned hot breads, pies, cakes, ice water, soda water, tobacco (chewed), large amounts of tea, coffee, and especially alcohol. Irregular eating is also a common sin. It has been said that the platter kills more than the sword, and that overeating produces a greater number of invalids than overdrinking, the soda fountains more than the saloon. Then in certain general diseases there is chronic gastritis, as in anæmia, tuberculosis, and Bright's disease. Local conditions, also, may bring about this trouble, as the irritation of an ulcer in the stomach wall, or the stagnation of food in a stomach with weak muscle walls which allow the food to remain there an unusual length of time and to decompose. And, lastly, there may be chronic gastritis in heart and liver troubles, and in other diseases which obstruct the circulation and allow the membrane of the stomach to remain turgid with dammed-back blood. Such a stomach wall does not get proper nourishment and, therefore, suffers.

The symptoms of chronic gastritis vary greatly. The appetite may be poor or good. There is usually some distress after eating, or "heart-burn" or "cardialgia." It is usually a little painful to press over the stomach; the tongue is coated; the taste in the mouth is bad; there is usually considerable nausea, and perhaps some vomiting, especially early in the morning—a vomitus which consists chiefly of mucus with the products of decomposition of the food; or the person belches considerable gas. If the gastric juice is examined chemically, there will be found little or no hydrochloric acid, and in extreme cases no gastric juice at all. Constipation is the rule. The patient is usually low-spirited and complains of dizziness.

The treatment of such cases is to remove the cause. This will not cure the condition, because the stomach has already suffered, but will lessen the symptoms. The person should eat only proper food, should chew it well, and, since the muscle of the stomach is weak, should eat small amounts and hence more frequently than normally. These persons are usually very unwise in their choice of foods; they eat what they want. A list of those things allowed should be written out, and the preparation of their food be supervised. It is often very wise to put them on a pure milk diet for a short while at least. Grains, vegetables, and the fats should be avoided; the hot bread, the pastry, and the fried foods should be omitted. If there is no gastric juice, or if the stomach is not able to digest the food, the patient can take hydrochloric acid or pepsin, or both. Dilute hydrochloric acid, from 15 to 100 drops at intervals after a meal, is far more important than pepsin, since it is the acid which disappears first. Usually there is enough of the ferments, but they cannot act because there is a lack of the acid. It is often desirable to wash out the stomach to keep it clean, and we may use water—a 1 per cent. salt solution, a 3 to 5 per cent. soda solution, or a 1:2000 silver-nitrate solution. The most valuable drug in such a case is really some bitter tonic, such as gentian, nux vomica, quassia, for these stimulate the secretion of gastric juice. The patient often gets relief by drinking large amounts of alkaline water at night or early in the morning, and some find it an advantage to take large amounts of salt. Above all else the psychic side is important, and whatever the patient eats

he should enjoy. Some, at least, of his food should be liquid in order to ensure a sufficient chemical stimulation of secretion. If the patient complains of a great deal of flatulency, it is well to avoid vegetables, starches, and fats. Among other drugs may be mentioned soda, bismuth, small doses of chloroform, and the compound spirits of ether.

DILATATION OF THE STOMACH.—The normal stomach has a capacity of about 1500 c.c.—about 3 pints—but the dilated

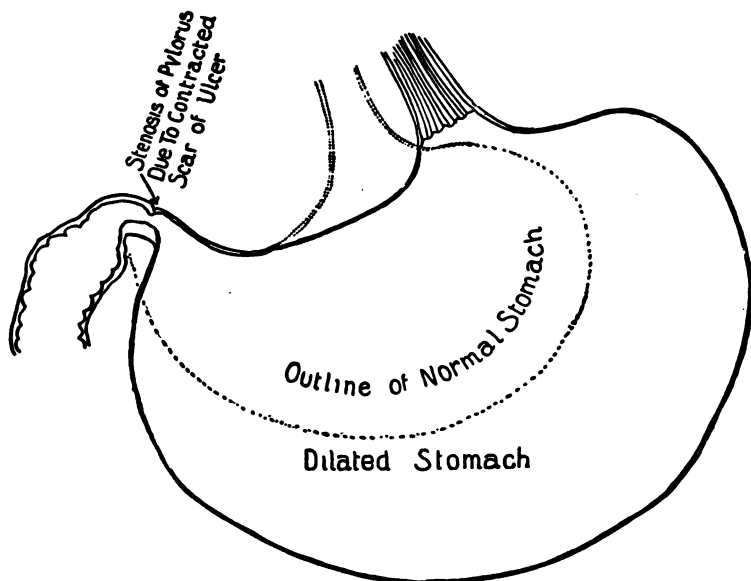


FIG. 53. Dilatation of the stomach due to the contracted scar of an ulcer.

stomach may hold even from 3 to 5 quarts. A patient with a stomach which for months does not secrete any gastric juice may be entirely unconscious of it, but one with a stomach even very slightly enlarged is soon painfully conscious of it, so important is its complete emptying. If food remains in it even a little too long there usually are symptoms, because of either the decomposition or the weight of the food.

Acute dilatation of the stomach occurs especially in acute fevers, such as pneumonia or typhoid fever. In these diseases it seems as if the poisons of the disease paralyzed the muscle wall.

Chronic dilatation is a very common condition. By this we mean not only a large but a weak organ. The stomach may be very large, but is not dilated if it can empty itself of the food within the normal time. If it can, no matter how large it may be, there will be no symptoms. Also, the stomach may seem large but not be dilated. There is a condition called "gastroptosis," which occurs especially in nervous women. Their stomach has descended to a much lower position than normal, the greater curvature has descended to the navel or below, and symptoms result. Sometimes not only the stomach but the liver, kidneys, and other organs have also "dropped down," a condition called "enteroptosis."

Chronic dilatation results first of all from some obstruction at the pylorus. There may have been there an ulcer which has healed, and the scar of which contracted. Sometimes this so constricts the pylorus that a lead pencil can hardly be pushed through the opening through which one ought easily to put three fingers. In other cases repeated spasms of the pylorus have made it hypertrophy so that it almost fills the aperture. A more common cause is a tumor which grows at this point. It may be small. It may be as large as a fist, but when well placed it may be small and yet very effectively stop up the pylorus. In other cases there is outside the stomach a tumor which is pressing on the pylorus. Other patients have had an inflammation near the pylorus—in the gall-bladder, for instance, and "adhesions" grow which may contract and almost close the pylorus, or may, by lifting up the pylorus, so kink it that the food can hardly pass through. In all these cases the trouble is at the pylorus, not in the stomach wall. The muscle of the wall must work unnaturally hard to force food out of the stomach, and the result is what one would expect; the muscle has more work to do, so that it gets stronger—that is, it hypertrophies, even to from 2 to 3 times the normal thickness. If we watch the patient two or three hours after a heavy meal, we can usually see the commotion going on. The vigorous contractions of the stomach can be easily watched.

The second cause is weakness in the stomach itself. The pylorus is normal, but the muscle is too weak to force the food through properly. This weakness may be the result of

habitually overfilling the stomach, or of the chronic inflammation of chronic gastritis; or it may be a part of a general weakness of the whole body, such as occurs in anæmia, tuberculosis, cancer, or rickets.

So long as the muscle is able to empty the stomach there may be no symptoms at all, but often there are nervous symptoms, and distress after eating. Sooner or later vomiting will begin. The patient may vomit only a small amount of food, but if the condition is of a fairly extreme grade he may vomit 3 or 4 litres, and in this vomitus can be recognized the food eaten two or even three meals before, or even three or four days before, and usually in putrid condition. This stagnant food injures the stomach wall, which soon secretes a gastric juice poor in quality and quantity. The food ferments or decomposes; it "sours" in the stomach. Since so much food is lost as vomitus, the patient loses weight and strength.

The diagnosis may be made from the vomitus, because, if this contains food eaten over seven hours before, the stomach is quite certainly dilated. If the person be made to drink a teaspoonful of soda dissolved in an ounce of water, and then another teaspoonful of tartaric acid in the same amount of water, the carbonic acid formed will inflate the stomach, which can then be seen to fill almost the whole abdomen.

For treatment, the person should improve his habits of eating, and, since the stomach has greater difficulty with a large than with a small meal, he should each day eat five small meals rather than three large meals. The food should be food easily digested. If the dilatation is severe the stomach should be kept empty by washing it out frequently, for if allowed to remain dilated it loses its tone more and more. If washed out regularly it may recover its strength, provided there is no great obstruction at the pylorus. The person should eat a pretty dry diet, and avoid such foods as ferment easily, such as fats, starches, vegetables. For medicines, the bitter tonics are to be recommended. In extreme cases surgical treatment is the only resource, and the pylorus can by simple operations be restored to its original size.

ULCER OF THE STOMACH.—By ulcer of the stomach we mean an excavation formed in the gastric wall by the death of a part of the mucous membrane. This hole may reach down to the muscle or even through to the peritoneum. It

is a question, which formerly was much discussed, why the stomach does not digest itself. Surely, the fact that it is living tissue does not protect it, for other live tissue can be digested if placed in the stomach. We cannot here discuss this question concerning living mucosa, but there is no doubt that if a portion of the wall is poorly supplied with blood, or dies for any reason whatever, that portion is quickly digested and an ulcer is left.

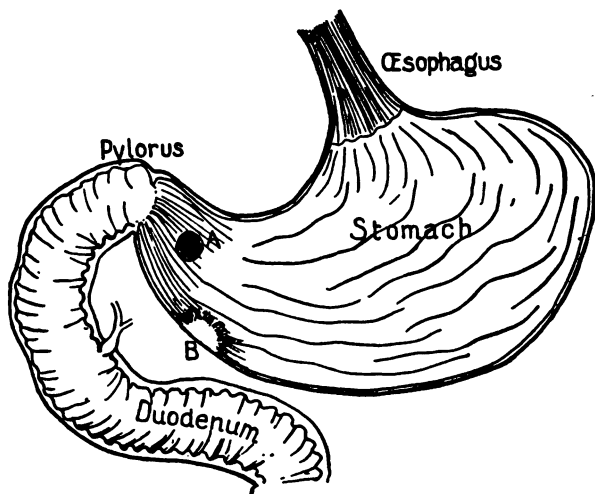


FIG. 54. Ulcers of the stomach. *A*, a small punctured-out ulcer near pylorus. *B*, a larger ulcer cut across.

Small superficial ulcers of the stomach are probably very common, do no harm, and rapidly heal over. The ulcers which make trouble seem to be those which are in a position—for instance, near the pylorus, that part of the stomach which moves most in peristalsis—where the continuous churning of the food and contraction of the stomach prevent healing.

Gastric ulcers occur in about 1 per cent. of all persons. It is strange that they should be more common in certain localities, as Great Britain and Massachusetts, than in others. Ulcers occur with greatest frequency between the ages of 15 and 25 years and in anæmic persons. They also occur in those persons whose work causes constant pressure on the stomach, such as the workman who always leans over the edge of a desk; the shoemaker, who presses the last against

the abdomen; and persons who constrict their waists too much by tight clothing.

As a rule ulcers are single, yet there may be many, even from 5 to 30. They occur most frequently near the pylorus, for the reason, perhaps, that in this position they have greater difficulty in healing. They may be small and clean, as if punched out by a sharp instrument, or they may be as large as the palm of the hand. Some have terraced edges. When they heal they leave a scar, as a rule, and this scar contracts with results which may be very serious if the ulcer was large; for near the pylorus (Fig. 53) it can almost shut off this aperture, and near the middle of the stomach it can contract the stomach considerably, giving rise to the so-called "hour-glass" stomach. The ulcer may completely perforate the stomach wall. If it does this slowly, the chances are that before there is an actual perforation the stomach will have become adherent to a neighboring organ—to the liver, for instance—and therefore little damage will result, but if this has not happened the stomach contents will pour through the perforation into the peritoneal cavity, and either a local abscess or a rapid general fatal peritonitis will be the result. The ulcer may open a large blood-vessel, with the result of fatal hemorrhage. In the floor of the ulcer the smaller arteries usually do remain patent, and since the wall here becomes weakened, a little aneurism about the size of a small bead forms, which often ruptures and gives rise to the hemorrhage which is so common in these cases.

The symptoms of ulcer are varied. The patient may have a large ulcer and yet never know it, but as a rule he will complain of dyspeptic symptoms, that is, of discomfort after eating. This discomfort amounts to actual pain in from 1 to 3 hours after a large meal, and this pain may be so great that the patient will roll on the floor in agony; or he may find relief by pressure, and will lean over the back of a chair or press a pillow against the stomach. There is usually a sore point which the patient can point to, and here even slight pressure is painful; even the skin itself is very sensitive. This pain after eating is probably explained by the irritation of the ulcer by the food. When one remembers that in churning the food the stomach normally uses considerable force, which scrapes the solid particles of food across the raw

surface of the ulcer, he wonders that the pain is not greater than it is.

About half the cases have hemorrhage; they suddenly vomit a large amount of blood—even a pint or a quart or more. The stools following this hemorrhage will be tar-like, because some blood will flow on into the intestine. The patient will often become very anæmic, almost exsanguined, and a most extreme secondary anæmia may result.

The third classical symptom is hyperacidity. In the majority of cases, if the gastric juice is examined chemically, too much hydrochloric acid will be found. Many think it is this high acid which is responsible for the formation of the ulcer.

The treatment is, of course, to allow the ulcer to heal, if possible. This may be done by complete rest in bed. One rests the stomach by feeding the patient by rectum, and by allowing only a little water or other bland fluids to enter the stomach. This rectal feeding may be continued for several weeks. During this time the ulcer, not irritated by food, and little irritated by contraction of the muscle wall, has a chance to heal over if it will. These patients, when they begin to eat, should be careful that their food is of such a nature that it will not form hard masses in the stomach. Milk is a very good food, although it can form hard clots which will rival pretty tough meats. Of all the meats, veal is most easily and quickly broken up into a soft mass, and hence is especially to be recommended. The reason for this is easily understood. In nearly all states there is a law that a calf shall not be killed for food until about three weeks after birth. Of course the farmer would prefer to kill the calf at birth, but the muscle of the very young calf, known at this age as "bob veal," is very soft and mucoid, and if eaten will cause considerable gastric distress, and diarrhoea. It does not taste like veal; in fact it was formerly canned as "chicken." This muscle later becomes hardened—that is, it becomes firmer, more like beef—and in about three weeks is suitable for food. But even then the mass of fibres is very easily broken up and rendered soft.

If the patient has had a severe hemorrhage, the anæmia, of course, will demand attention. In case there is suspicion of perforation, or the hemorrhage is very severe, it is much safer for the surgeons to operate, and to remove the ulcer.

CANCER OF THE STOMACH.—By cancer we mean, first, a new growth, a tumor. Just as a wart forms on the skin, is something which is not normally there, but is an enlargement of a tissue which was there before, so cancers are new growths of tissue. But cancers differ from other tumors in that the latter, the warts, cause trouble only where they grow, while fragments of cancer grow “wild,” that is, invade the neighboring tissues. Fragments, also, are carried through the body, and wherever they settle they are the starting-points

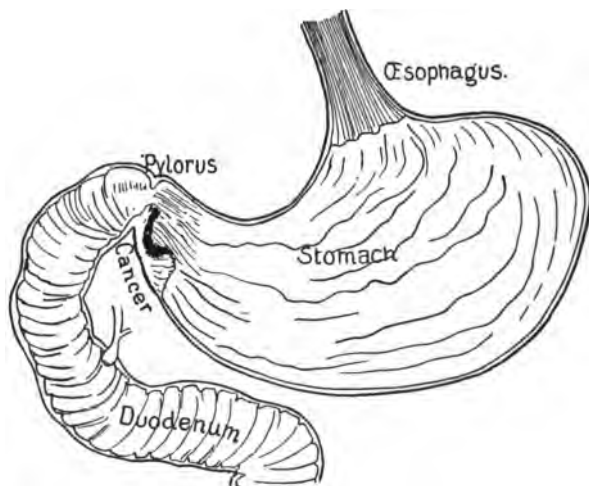


FIG. 55. Cancer of the stomach at the pylorus.

of new cancers, which are called “metastases.” Cancers, also, often slough very easily, so that what was a hard mass is soon an ulcer; but the well of the ulcer is the cancer. Cancer is a disease of middle age. It is very rare to find one in a person under 40 years of age.

The symptoms of cancer of the stomach are numerous. First, there are certain symptoms which are those of cancers anywhere in the body—that is, the loss of weight and of strength, and a progressive anæmia. Then we have special symptoms due to the presence of the cancer in the stomach, and the most important of these are sudden loss of appetite, and dyspepsia beginning at middle age. If two persons were to describe their dyspepsia in just the same words, but one

were to say that his trouble began when he was a young man and had been present more or less ever since, while the other were to declare that he had had a normal digestion until he was forty years old or more, we might be reasonably sure that the former had no cancer, while very possibly the other has. If in addition to this suddenly developing dyspepsia at middle age he has had also a loss of weight and strength, and has become rather pale, we may be pretty sure of our diagnosis.

Then there is the vomiting of coffee-ground vomitus. The blood which slowly leaks from the cancer becomes partially digested and therefore of a dark coffee color. Large hemorrhages are very rare. The patient may not vomit, but we can easily find this blood in the stools, if we examine them carefully. Then the patient may complain of pain in the stomach.

If we examine the gastric juice, we shall find that very early there is no hydrochloric acid present, but some lactic acid formed from the gastric contents. We may be able to feel the tumor. We may find other tumors, the metastases, especially on the surface of the liver or in the skin at the umbilicus.

The third group of symptoms of cancer of the stomach consists of those which depend on the exact location of the cancer in the stomach. If it is near the pylorus, it will cause an obstruction there, and then all the symptoms described under dilated stomach will also be present. If it is at the cardiac orifice, the vomiting will immediately follow the meal.

The stomach will also, because of the stagnant contents, soon be the seat of chronic gastritis, and all symptoms of this condition will be added to those of cancer. Lastly, we have a certain number of patients who have no symptoms at all, but, dying for some unknown reason, are found to have a cancer in the stomach.

The treatment for very early cancers is surgical, for it is possible that sometimes the disease may be entirely removed and the patient get well, or, at any rate, have several years of comfort added to his life. It is true that he may die of cancer in from two to five years after the operation, but the chances are that during these years he will be fairly comfortable, for the most of the distress a patient with gastric cancer

complains of is due to his dilated stomach. Then, the patient with even a large cancer may be made comfortable by proper diet, by keeping the stomach well washed out, etc. When the pylorus is closed the intestine may be sewed to the stomach and a new pylorus made.

Neuroses of the Stomach.—**NERVOUS DYSPEPSIA.**—Many persons suffer intensely from “dyspepsia,” whose stomachs are, so far as we can tell, without any trace of disease. The stomach is “all right,” but it doesn’t work right. Many persons refuse to believe that stomachs which pain them so are “all right.” The man who is rolling about on the floor, screaming with pain, and begging for any operation which may cure him, is skeptical as to his stomach’s being “all right.” Of course we exclude cases of gall-stones, etc. When we know that a stomach has not secreted any gastric juice for five years it is hard to believe that this stomach is “all right;” the person who vomits so severely that he loses weight and strength, and thinks he is at the point of death, is slow to believe the trouble is not in his stomach; and yet such is often the case.

The neuroses of the stomach may take one or more of three forms; motor, secretory, and sensory. When one sees the stomach at work, realizes how powerful are its muscular contractions during normal peristalsis, notes that food is poorly chewed and contains hard masses of vegetables, seeds, bones, and tough fibres, and remembers that these vigorous muscular movements scrape these hard masses over the delicate mucous membrane, he can only wonder that all normal digestion is not exceedingly painful.

To a great extent the stomach, its secretion, the act of vomiting, etc., are governed by the nervous system—by organs at some distance. The real source of some of the most violent gastric symptoms is not in the stomach at all, but is in the nervous system. This is beautifully illustrated by the gastric crises of locomotor ataxia. These patients have violent attacks of vomiting, etc., yet the stomach is perfectly normal. Its nervous control is much disturbed. It behaves much as a steamship would were the officer on the bridge to give the engineer a succession of crazy orders. In very many cases the nervous trouble is not organic, as is locomotor ataxia, but purely functional. The result is a “gastric neuro-

sis." The person is nervous, neurasthenic, "run down," and shows this by his gastric symptoms. His stomach has faulty nervous control. In most cases the gastric symptoms are only a small part of a large number of nervous symptoms, but in other cases the general neurosis is so overshadowed by the severe stomach symptoms that we do not suspect the person to be hysterical or neurotic.

In a large group of cases there is real trouble, not in the stomach or in the nervous system, but in some distant organs with which the stomach is connected through the nerves. For instance, in chronic appendicitis, intestinal obstruction, and some other intestinal troubles, the person has bad attacks of vomiting. It would seem as if the intestine were trying to protect itself from the food eaten by telephoning the stomach through the nervous system to vomit, and thus prevent this food's reaching the intestine and irritating the disturbed bowel. Other troubles, such as pelvic diseases, also produce symptoms in a normal stomach. In other cases "functional disturbances" follow years of abuse of the stomach, and the only wonder is that more do not suffer. So many persons abuse their stomachs so abominably. Some eat at very irregular hours; some habitually eat too much; some eat improper food; while more do not masticate their food sufficiently, or go to hard work at once after eating.

A person in normal health knows nothing of the commotion in his stomach; he is entirely oblivious of it. And yet there must be a continuous stream of sensation flowing from stomach to brain, which never reaches consciousness. If, now, for any reason one becomes nervous and pays attention to these sensations, many messages to the brain are felt and even amount to pain, which formerly would not have reached consciousness. We cannot blame persons who suffer in this way; their pain is intense. It is only unfortunate that they cannot suppress these stimuli below the threshold of consciousness, as can the normal person, without effort.

Among the MOTOR NEUROSES is, first, supermotility. A stomach thus affected empties itself before the food is properly digested. There may be no symptoms, and the condition may be learned only by removing meals at stated intervals. Other nervous persons have a condition known as "peristaltic unrest." After a meal they can feel the peristalsis, and the

stomach often makes loud rumbling which can be heard at some distance. "Aërophagy," or "air swallowing," is another motor trick. The horses which do this are called "crib suckers." It is not an unusual symptom in nervous persons. It may come on in attacks lasting hours or even days. These persons belch large amounts of air with noise enough to be heard at some distance. One wonders where the air comes from, but there is little doubt that they are continually swallowing the air, and then, as it were, seeing how much noise they can make in belching it. "Nervous vomiting" is also a motor neurosis. Vomiting itself is not a gastric symptom alone; the nervous system does the most. There are some patients who vomit either occasionally or very frequently—some who vomit so often that they starve to death; and yet their entire trouble is a neurosis. Among these patients are some who vomit without nausea. They regurgitate their food and then spit it out. Sometimes this is a trick which a child learns and practices. In one recent case, a very attractive, even prominent, young woman had practiced this as an accomplishment when a young girl fourteen or sixteen years old, and had taught it to her friends. They had contests to see who could vomit the most. As her friends grew older they were sensible enough to break the habit, but in the case of this patient the habit was not so easily broken. "Rumination," or "chewing the cud"—that is, regurgitating the food from the stomach and chewing it over again—is a trick of some hysterical persons, but more commonly of those mentally deficient.

"Cardiac spasms," or painful contracture of the muscle which surrounds the orifice connecting the œsophagus with the stomach, is sometimes an hysterical symptom. Sometimes it is caused by too hot or too cold food or by too rapid eating. "Pyloric spasm" is a more common symptom. In this case the muscle closing the orifice between the stomach and bowel contracts vigorously and painfully at the time when the stomach is forcibly contracting in order to force the food on, and the result is as severe a pain as is found in any true stomach disease. This may be a purely nervous symptom, but is more apt to be due to the presence in the pylorus of a small ulcer, which is irritated by the passage of food or by a too acid gastric juice; or to gall-stones, which reflexly stimulate the pylorus.

Another motor neurosis causes atony of the stomach. It will not contract well, and therefore becomes distended with the food—overstretched, as it were—and all the symptoms of moderate dilatation of the stomach can result.

SECRETORY NEUROSES are very common. The gastric juice secreted during digestion contains normally about 0.2 per cent. of hydrochloric acid, but may contain twice or three times as much acid as this, a condition called “hyperacidity.” Persons with hyperacidity complain of pain which begins from one to three hours after a meal, or when secretion is most active. There is a sensation of weight or pressure, and the person belches up a good deal of sour, irritating, acid juice. Whether a person suffers from too acid gastric juice depends quite as much on him as on the juice; we repeatedly find normal persons who are unconscious that their gastric juice is more acid than the worst of these patients, and, others, again, who complain of the most severe symptoms, and yet on examination are found to have a gastric juice which is no more acid than normal.

Hypersecretion is a more common condition. By this we mean that too much gastric juice is secreted and usually all the time, both when there is food in the stomach and when it is empty. In a great many cases this is not a pure neurosis, but the stomach is slightly enlarged and always contains fragments of the food, which act as a constant stimulus to secretion. In these cases the condition lasts for a long time. In a truly nervous case the hypersecretion comes on suddenly, lasts for a few hours or a few days, and then just as suddenly disappears. The patient has a sudden fright, or is excited or worried, or hears bad news. Almost at once he feels a gnawing pain in the stomach, his head begins to ache, he then begins to vomit large amounts of a clear gastric juice which is very burning and irritating, both in the stomach and in the mouth. He vomits incessantly, but especially at night and early morning. The best example of this trouble is not in a pure neurosis but in locomotor ataxia, a fatal nervous disease, yet a condition in which the stomach is not diseased.

Subacidity—that is, the secretion of too little gastric juice—is usually due to severe disease of the stomach, but may be a purely nervous condition. In some cases no juice at all is secreted. This is termed “anacidity.” One stomach

which had secreted no juice for five years all of a sudden began to behave normally.

SENSORY NEUROSES.—Sensations from the stomach are usually present with all the other neuroses, but the one wonder is that a normal person is not painfully aware of the processes of digestion. There are some persons who feel intense discomfort during the digestion of each meal; there are others who are distressed by certain articles of food, and that this is simply a nervous symptom can be seen from the fact that if you prescribe that same food as a medicine it will cause no pain, but let them eat it as a food and the pain is intense. The feeling in this case is of fullness, weight, and burning.

By *gastralgia* is meant severe paroxysms of pain. This pain may be agonizing, and even rival or surpass in intensity the pain of gall-stones, liver, or true gastric diseases. It can simulate the pain of these conditions even to the minutest detail, and yet, when we operate and examine the stomach and liver, we find everything is perfectly normal. These cases are very frequently operated on. They insist upon it, and in some cases the operation cures the nervous condition, but more frequently it does not, especially if the patient is told that the stomach was found to be normal.

CHAPTER V

DISEASES OF THE INTESTINE

The Intestine is a muscular tube which begins at the pylorus (Fig. 51, *b*) and ends at the anus. The first twelve inches from the pylorus are called the duodenum (Figs. 52, 54, and 55); into this part enter the gall-duct (Fig. 52, *c*) and the pancreatic duct (Fig. 52, *d*). The small intestine is about twenty feet in length. The upper two-fifths below the duodenum are the "jejunum," the lower three-fifths are the "ileum." The ileum opens into the colon, or large bowel. (Figs. 51 and 61.) Just below the opening of the ileum,—an opening which is protected by a valve, the "ileo-cæcal valve,"—is the "appendix vermiformis." The large bowel is about five feet long. It first ascends to the liver, as the "ascending colon," then across the abdomen, as the "transverse colon;" it then descends, as the "descending colon," into the left flank. The next part is very tortuous, is named the "sigmoid flexure," and passes into the "rectum."

The stomach and intestines work in harmony in the digestion of food. In the stomach the food is stored up and is gradually reduced to a liquid, or at least to a very soft pulp, which is doled out to the intestine as rapidly as that organ can accommodate it. The stomach begins digestion, the intestine ends it. The food is not absorbed in the stomach—that is the chief function of the intestine. Here it is that the food passes into the blood and lymph vessels and is transported to the various parts of the body.

Into the duodenum opens the pancreatic duct (Fig. 52, *d* and Fig. 73), through which pours the pancreatic juice. This duct and the common bile duct unite, and open through a common orifice at a point eight or ten centimetres below the pylorus. Between 500 and 800 c.c. of the pancreatic juice are secreted every day. This juice is an intensely powerful digestive fluid. It contains three ferments—trypsin, diastase and steapsin. Trypsin is the most powerful ferment known. Its

action is similar to that of pepsin, in that it breaks the protein molecule to simpler and simpler bodies; but it is infinitely more powerful than pepsin, breaks the protein down to much simpler substances, and does it with much greater rapidity. So powerful is trypsin that there is little wonder that the body protects itself by manufacturing it as trypsinogen. That is, the pancreatic juice as it flows through the duct cannot digest protein at all; but let it come into contact with the mucous membrane of the intestine, and harmless trypsinogen is changed at once into the powerful trypsin. This is due to enterokinase, a ferment in this mucosa, which "activates" the trypsinogen. Another difference between trypsin and pepsin is that while pepsin can act only in an acid juice, trypsin requires an alkaline medium. Amylopsin, or diastase, is practically the same as the ptyalin of the saliva. That is, it breaks the complicated sugars down to the simple sugars—glucose, levulose, etc. Lipase, or steapsin, is a ferment which splits fat into its two components, glycerin and fatty acids. The pancreatic juice sometimes contains other ferments. In children it contains lactase, which is important in digesting milk sugar. The pancreatic juice is also very alkaline and hence changes the acid food from the stomach into an alkaline food and furnishes the alkali which with the fatty acids makes soap.

The bile also enters the intestine at this point (Fig. 52, c). The greatest part of the constituents of bile are substances which are no longer of use to the body. It contains also some alkalis which help to neutralize the acid from the stomach, and some substances in which soaps and the fats are dissolved, and which aid much in the absorption of fat in the intestine.

Along the whole length of the small intestine the mucous membrane secretes small amounts of "intestinal juice," which is also valuable in digestion. This juice completes the digestion of the protein, and helps split the fat, but one of its most important constituents is "invertin," a ferment which is important in carbohydrate digestion. Ptyalin and amylopsin split the complex carbohydrates into simple sugars, but invertin "inverts," or changes, all the simple sugars into glucose.

In review, then, water and salts are not digested. Water does not stay more than one or two minutes in the stomach,

but is passed on into the bowels and there is absorbed. The *salts* also are absorbed here. The digestion of *carbohydrate* begins in the mouth, continues in the stomach until the gastric juice is so acid that the *ptyalin* is destroyed, and is continued by the pancreatic juice. All the complex carbohydrates—starches, sugars, etc.,—are first broken up into simple sugars, and these the *invertin* transforms to glucose, the only form of sugar of much use to the body. Fats are split in the stomach to a limited degree, but for the most part this is done by the pancreatic juice, which also furnishes the alkali that changes the free fatty acid into soap. The soap and the glycerin are then absorbed. The digestion of protein begins in the stomach. The *pepsin* breaks up the higher proteins into the simpler bodies, such as *peptone*, etc. *Trypsin* quickly breaks these up into much simpler bodies. The process by which all this is done is called “hydrolysis;” that is, a molecule of water is the wedge and the ferment the mallet by which these huge molecules are split into smaller ones. There are some proteins which *pepsin* cannot attack at all. These *trypsin* digests. There are other proteins which *trypsin* cannot digest unless *pepsin* has already begun the splitting. There are many which *trypsin* could digest, but it does it better when *pepsin* has already begun the process. Thus we can say that gastric and pancreatic digestion work together. The *pepsin* prepares the way for the *trypsin*.

Bile flows into the intestine quite continuously, but the flow is greatest after a meal. Pancreatic juice is not secreted while a person is fasting. When, however, food reaches the stomach, then the flow of pancreatic juice begins. It reaches its maximum about three hours after the meal, the time when most of the food is being poured into the intestine, and then slowly diminishes as the food is gradually digested. It is of interest that the stimulation for pancreatic secretion is chiefly chemical. A substance called *secretin* is formed in the stomach and intestine, is absorbed in the blood, carried to the pancreas, and there forces this organ to secrete pancreatic juice. We can, therefore, make the pancreas work at any time when we wish by injecting into the blood of an animal or person a little *secretin* from another animal.

The composition of gastric juice is not always the same. In fact, the gastric juice varies according to the task before it.

So it is with the pancreas. The amount, the quality, and the quantity of the pancreatic juice after each meal depend chiefly on the food it must digest. A very pretty illustration of this is seen in the digestion of milk. In young animals on a milk diet, the pancreatic juice contains lactase, a ferment quite important in the digestion of milk. After the person has ceased to drink much milk, this lactase disappears. Many adults complain that milk distresses them considerably. The trouble with them is partly the absence of lactase. Since it is very necessary in the treatment of nervous diseases that patients should drink large amounts of milk, it is well to give for a few days nothing but milk, and in small amounts increasing each day for about a week. At the end of that time the pancreas has accustomed itself to the task before it and furnishes enough lactase. A person can now easily digest five or six quarts of milk a day, and without a single symptom. So it is with all our meals.

The gastric and pancreatic juices are modified to meet the requirements of each meal. The intestinal fluid contains four or five ferments. Enterokinase has already been mentioned as the one which activates the trypsinogen, and secretin as the substance which stimulates the pancreas to secrete its juice. There are inverting enzymes for each sugar—maltase to invert maltose, invertase to invert cane sugar into dextrose and levulose, lactase to change milk sugar into dextrose and lactose. Erypsin is a powerful intestinal ferment whose function seems to be to destroy that protein which has escaped absorption.

The function of the small intestine is to complete digestion and to absorb the food, which is now in fluid condition. The mucous membrane lining the wall of the small bowel is thrown into folds to increase its surface (Fig. 56), and not only that, but has a surface much like velvet, being completely covered with finger-like processes known as villi (see Fig. 56), which vastly increase the absorbing surface.

By the time the food reaches the ileo-cæcal valve, practically all that is of value has been absorbed. That which is left is the indigestible part of the food, especially cellulose, together with the constituents of the bile and intestinal fluids, a great many old epithelial cells (for the mucous membrane of the intestine, like the external skin, is constantly

throwing off its used-up cells), and vast numbers of the bacteria, which grow in the intestine.

After a heavy meal the first of this refuse reaches the ileo-cæcal valve in from three to five hours, and the last in from nine to twenty-three hours. In the large intestine this fecal matter very slowly moves towards the rectum. It enters the colon mixed with much fluid. Here the water is absorbed, and the intestinal contents are gradually dried down to a solid mass. In the small intestine are many bacteria, chiefly the milk sourers (*Bacillus lactis aërogenes*), but in the large intestine *Bacillus coli communis*, usually called the "colon

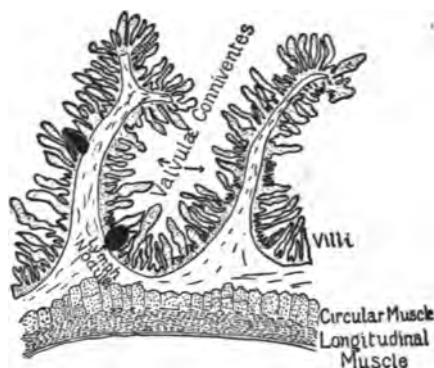


FIG. 56. A section through the wall of the small intestine (jejunum) made parallel to the length of the bowel, showing the folds of the mucosa, and the villi on these folds. (Magnified.)

bacillus," predominates. When evacuated, perhaps one-fifth or more of the solid stool is bacteria. The function of these bacteria nobody knows. Many think that they are a benefit to us, for it has been shown that animals do not do as well if brought up from birth in such a way that bacteria cannot enter the intestine. In the large intestine they seem to help putrefy whatever food may have escaped digestion.

DIARRHŒA.—By diarrhœa one means the passage of too frequent and too soft stools. As has been said above, the intestinal contents enter the large intestine for the most part as a fluid, and the function of the large intestine is to absorb the water. The stools will therefore be abnormally soft if the peristalsis of the small or of the large intestine is so rapid that there is not time to absorb the water, or if disease of

the bowel wall prevents this absorption, or if fluid is actually added from the blood to the intestinal contents. The latter occurs in many bacterial infections of the intestine, but the best illustration is Asiatic cholera, a disease in which the fluid of the stools comes for the most part from the blood (see page 293). Diarrhœa may be caused by food. The patient may have eaten too much food, or not chewed it enough, or the food may be unsuitable, as unripe fruit, which cannot be digested, and over-ripe fruit, which decomposes rapidly. In these cases the food is an irritant and causes irritation and increased peristalsis of the bowels. Then, increased peristalsis may be caused by true poisons, such as arsenic, mercury, etc. Changes in the weather will give some persons diarrhœa, especially if the weather changes suddenly to cold. There are some foods which are poisonous to certain persons and always give them diarrhœa.

In many cases, however, the trouble is not in the food or the intestine, but in the mind of the person; for frequency of stools and rapidity of peristalsis are in some measure controlled by the nervous system. There are some persons with a nervous diarrhœa; that is, the diarrhœa is the result of abnormal nervous control of the bowel. Some persons with this "nervous diarrhœa" have after breakfast one or two loose movements. They are sure that the food which they have just eaten passes through at once undigested. This is of course not the case. Some have one or two fluid movements after any mental excitement or emotion, and there are some whose bowels are pretty apt to move when it is most inconvenient. In all these cases it is the nervous control of those functions that is at fault. Certain infectious diseases are accompanied by diarrhœa, as typhoid fever and tuberculosis. Here it is probably the actual disease of the bowel wall that explains the diarrhœa. In heart and liver diseases with chronic passive congestion of the bowel wall (see page 61) there is often diarrhœa, while other persons, when run down, have a diarrhœa.

Diarrhœa may be acute or chronic. If acute, following some sudden onset, it will usually be accompanied by vomiting and colicky pains. If the small intestine is at fault, lientery, or the diarrhœa of undigested food, is often present.

Many persons do not understand the difference between

diarrhoea and dysentery. Diarrhoea is a symptom. The patient has too frequent and too fluid movements. By dysentery is meant an actual inflammation of the bowel wall, shown clinically by the blood, pus, and mucus in the stools. Some of the stools contain only these substances and no food at all. In simple diarrhoea the movements are usually large and not very frequent, whereas in dysentery they are very frequent, are scanty and are passed with a great deal of pain ("tenesmus").

In the treatment of diarrhoea the cause is to be considered. If the diarrhoea is acute, the patient is better off in bed and on a diet of boiled milk. Since the trouble is often due to some irritating substance in the bowel it is very desirable to give a purge, preferably calomel, to remove this. Bismuth in very large doses is satisfactory, for it seems to have a soothing effect upon the bowel. If necessary, a little laudanum or a lead and opium pill, one grain of each, is given. In all cases of chronic diarrhoea, it is very necessary to seek the cause. This may be an intestinal parasite, such as the hook-worm, or some form of dysentery, as amœbic dysentery, or tuberculosis with ulceration. A simple diarrhoea may be due to as serious a condition as cancer in the rectum. In the case of women it is often due to trouble in the pelvis. These troubles of course need treatment. In very many cases, however, diarrhoea is simply a nervous phenomenon, and for that the general treatment of neurasthenia, with no local treatment, is indicated.

ULCERS OF THE INTESTINE.—The small bowel is ulcerated in two diseases especially, in typhoid fever and in tuberculosis of the intestines. The symptoms in these cases are those of the general disease. The large bowel is ulcerated in the various forms of dysentery and in tuberculosis.

CONSTIPATION is an abnormal infrequency of stools. But there is no absolute standard for this other than the subjective symptoms of the patient. Most persons have one or two movements a day. But, while some are very uncomfortable if for any reason they do not have one, others habitually go two, three, or four days without a movement and suffer no discomfort.

One point it is important to remember, that a person cannot judge of constipation simply by the number of move-

ments he has; the subjective symptoms also count. Some persons have regularly one large movement every day and would scoff at the idea that they were constipated, and yet they suffer from the symptoms of constipation. Their trouble may be "latent constipation." That is, the stool of one morning is not the refuse of the food eaten the day before, but of that eaten two or three days before; their bowels contain the food of two or three days, and the absorption from this stagnant mass can cause serious symptoms. Again, there is an old French adage that "diarrhoea is the best symptom of constipation," and this is actually true. Some very constipated persons have in their colons masses of very hard, dry stools, which cling to the wall of the intestine. There they set up an irritation, so that there is a diarrhoea of liquid stools around them. Such a patient will have alternating periods of constipation and diarrhoea. In some at least of their fluid stools will be seen small, very hard lumps of faeces. If the food remains long in the colon practically all of its water will be absorbed, and the masses may be almost as hard as a stone. These large fecal masses usually crumble into smaller masses, giving the condition known as "sheep faeces." One or two of these in a diarrhoea stool will at once give us a clew to the diagnosis. The lower bowel may in a severe case of constipation become "impacted"—that is, filled by a mass of hard faeces which must be removed by the fingers, or at first softened by large injections of oil.

The causes of constipation are many. Among the members of some families it seems almost a normal condition. It is most common in persons of sedentary habits, for a certain amount of muscular exercise is necessary for the proper functioning of the bowels. One of the most important causes, however, is the food, and it would seem as if our bowels were made to suffer through what we consider advances and refinements in our life, especially in our diet. For instance, for the proper functioning of the bowels they should contain a large amount of residue; but the finer and whiter the flour, the less of the wheat cellulose it contains. We need to eat a lot of "husks" and "fibres" and indigestible stuff in general. Persons troubled with constipation should eat much of the foods containing "sticks and stones and strings and bones." Another cause for constipation, which applies especially to

women, is weakness of the abdominal wall. A very important cause is habit. Many busy persons do not observe regularity in this particular and suffer considerably. These are the patients who assure us that for nine, ten, or even twelve years they have taken laxatives practically every day. They have no excuse for their condition, and in three or four weeks could break this habit and remain regular if they should really try. Other more serious causes of constipation are liver disease, intestinal disease, acute fevers, the morphia habit, dysentery, etc.

The symptoms arising from constipation are very varied. Some persons have none at all, even after a week of constipation, but most have a feeling of lassitude, headache, mental depression, or loss of appetite, and in some it causes symptoms of very serious diseases.

It is constipation that gives the patent-medicine dealers their best customers. When the label on the bottle states that the contents can cure, "liver disease, stomach disease, intestinal disorders, kidney disease, heart disease, brain disease, melancholia, in fact can make you young again," the chances are that this medicine is a simple laxative which can be taken for a long period of time; for chronic constipation can cause some of the *symptoms* of all these diseases, and some symptoms of greater severity than do the diseases themselves. Biliousness means constipation much oftener than it means liver trouble, and as for the last promise on the label, it is certainly true, for some of the most cantankerous, melancholic, pessimistic, bad-tempered hypochondriacs become cheerful, optimistic citizens after their constipation is cured. Constipated persons seem to have an auto-intoxication, by which we mean a poisoning of the whole body by the absorption from the mass of decomposing, stagnant fæces. Diarrhoea as has already been mentioned, is also a symptom of constipation. The large, hard, fecal masses in the colon often cause neuralgia of the sacral nerves; while one of the commonest results of constipation is hemorrhoids, or "piles."

To cure chronic constipation we must of course remove the cause. The person should eat food containing considerable residue, as whole-wheat bread, cereals, fruits, spinach, carrots, asparagus, celery, lettuce, turnips, parsnips, that is, those vegetables which are stringy in nature. He should eat also

certain foods which stimulate the peristalsis of the intestines, such as sugars, honey, marmalade, etc. It will be seen from this list that the *menu* of the breakfast of the average family has especial reference to the needs of the bowels. For breakfast many persons eat fruit, then a cereal, then honey or marmalade with bread and butter. Many eat nothing else for this meal, and none of these articles of food at either of the other two meals.

The most important element in the cure is the acquiring, by the patient, of that habit which every person should have, of perfect regularity in the movements of the bowels. The patient is told to try to have a movement at a certain convenient time every morning in the year. If he chooses nine o'clock in the morning, it should always be nine o'clock, not five minutes before or five minutes after, at least during the time when he is in training. He should daily at that time try, and try hard, to have a movement. If he succeeds, well and good. If he fails, he should try not to have a movement until nine o'clock of the next day. In this manner he acquires a habit. Some severe cases state at the end of three or four weeks that they can tell without looking at the watch when it is five minutes before nine o'clock.

In some cases a little treatment is necessary—a little cascara sagrada or a glycerin suppository. Glycerin is of value because it makes a movement easier, and, more important, it irritates the bowels. Normally, whenever fecal matter is in the rectum disagreeable sensations warn the person that the rectum needs emptying. But a man can get used to these sensations as to any others and then they will cease. Very often it is advantageous to soften the movement by a large amount of water or oil, but it is customary to add a little soft soap to the water, and glycerin to the oil, in order to stimulate the bowel.

Often in acute constipation some drug is necessary. The list of purgatives is of course long, and each has its advantages. There is, however, one point to be emphasized, that in the great majority of cases the trouble is with the last two or three feet of the large bowel, and it is foolish, often even injurious, to take a drug which is going to affect twenty-five feet of bowel in order to reach the last two or three feet. Fortunately, we have drugs, such as aloes and cascara, which

stimulate the great bowel especially. Of course in the acute constipation which accompanies acute fevers, tonsillitis, a severe cold, etc., it is necessary to clean out the whole of the small bowel, and then calomel or salts may be best. In the severe constipation of Bright's disease elaterium, and even croton oil may be necessary.

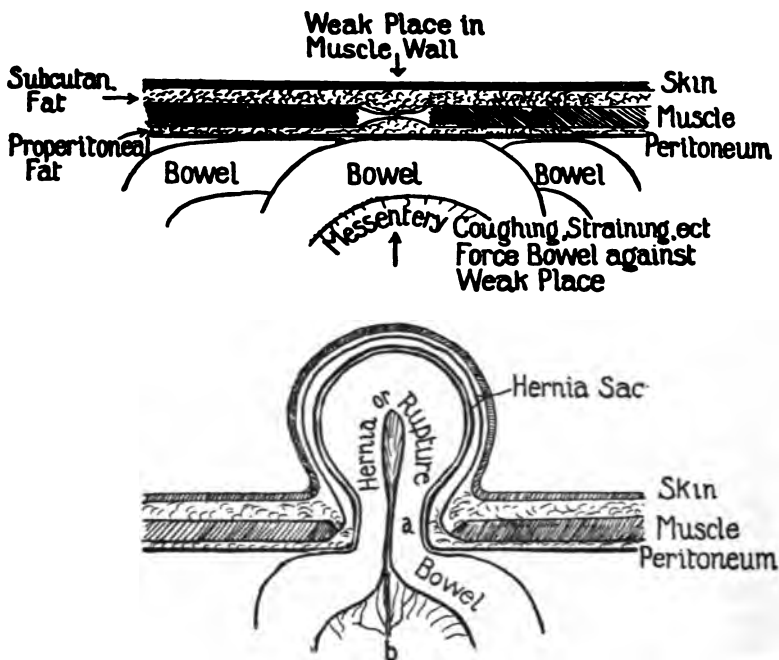


FIG. 57. Hernia or "rupture." The weak place in the muscle wall pictured above might be any of the "rings," or points weakened by the passage through the muscle of blood-vessels, etc. *a*, is the neck of the sac, the point where the bowel is pinched by the ring of muscle; *b*, is the artery supplying the loop of bowel with blood, and of course occluded if the pressure of the ring is great.

INTESTINAL OBSTRUCTION.—By intestinal obstruction is meant any condition which prevents intestinal contents from flowing through the bowel. This usually is serious, not because the bowel contents are dammed back, but because there is usually also some obstruction to the circulation of the bowel wall; and this usually means, sooner or later, peritonitis.

Intestinal obstruction may be due to a tumor, usually a cancer, growing inside and gradually filling up the bowel.

A chronic and severe constipation may be almost the only bowel symptom of such a trouble. Or the obstruction may be due to a foreign body lodged in the bowel, such as false teeth or fruit stones, etc. In other cases the bowel is "pinched" by the contraction of the scar of a healed ulcer in its wall. In none of these cases is there much immediate danger.

Hernia, or *rupture*, is one of the most common and important causes of intestinal obstruction. By this we mean that the bowel has forced itself through one of the weak points in the abdominal wall, such as the navel, the femoral rings, or the inguinal rings (see Fig. 57). There are also pockets of peritoneum in the posterior wall of the abdomen,

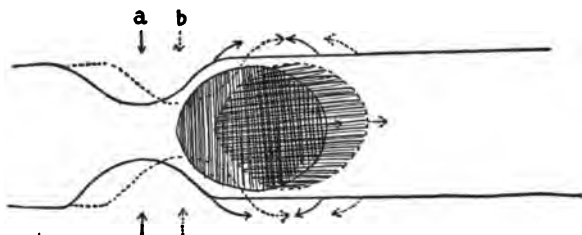


FIG. 58. Diagram of a peristaltic wave. In the bowel is a bolus of food. The entire line, *a*, represents the beginning of the wave; the dotted line, *b*, the wave a moment later.

and in these the bowel may get caught. The bowel is in constant peristaltic movements. The muscle fibres in rings around the bowel contract, and this constricts the lumen of the bowel (see Fig. 58); the fibres which run parallel to the lumen contract, and this shortens the length of the loop. The waves of these two movements pass in orderly succession down the bowel. It is an interesting fact that if a loop gets caught in a small opening, the rest of the bowel, because of the peristaltic waves, will try to follow it in. We may find almost the whole bowel in one of these pockets, or through one of these rings. The patient usually can push it back easily, but sometimes it gets pinched in the opening, and he cannot push it back. Then the hernia is said to be "strangulated"—that is, so tightly pinched that its circulation is stopped (see Fig. 57). In such a case the bowel wall is sure to die, and the result is a fatal peritonitis.

Intussusception is a very common cause of intestinal obstruction, especially in children. By this we mean that the

bowel above a certain point pushes itself into the bowel below that point (Fig. 59), much as a telescope is made shorter by pushing one section into the next, or as the finger of a glove may be pushed down into the hand. This also is due to the peristaltic waves. The most common point for this to occur at is the ileo-cæcal valve (Fig. 51 and Fig. 61), and one may sometimes see the small bowel protruding at the anus after making its way down the whole length of the large bowel. Any child who has been seized with pain in the stomach and then passes blood and mucus in his stools should be examined to see whether or not he has an intussusception.



FIG. 59. Intussusception. The bowel is represented working its way down within itself. Of course the mesentery containing the blood-vessels is dragged down also and the vessels closed by pressure resulting in the death of the inner portion of the loop contained within the outer portion of this loop.

And, lastly, *knots* and *twists* of the bowel may obstruct it. A loop may become twisted about itself or may tie itself into a true knot (Fig. 60). Here also peristaltic motion is to blame.

The symptoms of intestinal obstruction are important to recognize. There is always perfect constipation. The patient may pass blood and mucus in the stools, but no fecal matter. The patient's vomiting is often peculiar and unmistakable. When there is a true obstruction the peristaltic waves become very vigorous, but in the reverse direction; this is known as "antiperistalsis." All the intestinal contents are now moved toward the mouth, just as previously they were moved towards the rectum. This patient vomits first the contents of the stomach, then the bile-stained contents of the upper small intestine, then the darker, worse-smelling, intestinal contents of the ileum; and finally, if the obstruction is in the large intestine, the true fecal masses with their dark appearance and their foul odor will be vomited. This is known as "fecal

vomiting." The nature of the vomitus will depend on the position of the obstruction. Only when it is in the colon can feces be vomited.

There is always pain, at first sharp and colicky, and later continuous. Soon the patient may show collapse and then follows death. If the obstruction completely stops the circulation of the loop, that loop of bowel dies and becomes gangrenous, and a fatal peritonitis sets in. In chronic obstruction the symptoms are much slower in their development.

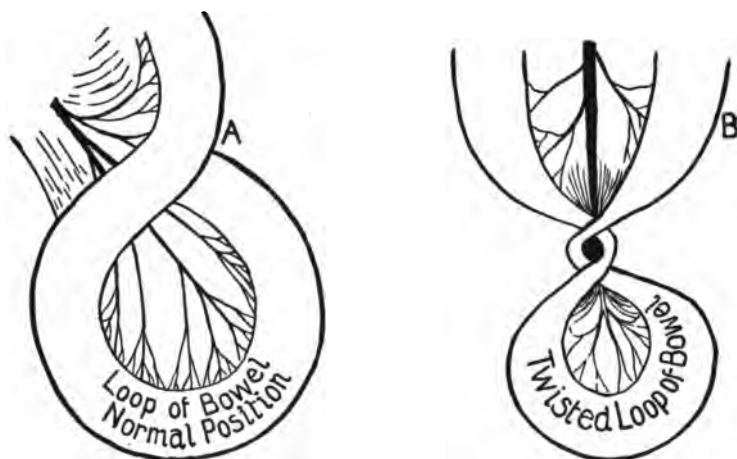


FIG. 60. Intestinal obstruction caused by a twist of a loop. A, the normal position of a loop. B, the result of a twist. This will occlude the bowel and also close by pressure the blood-vessels in the mesentery, causing the death of the bowel beyond the twist.

The constipation may last for days, the abdomen become very much swollen, and fecal vomiting occur, but since there is no obstruction to circulation in the bowel wall, the symptoms of peritonitis are absent or appear very late.

The treatment of intestinal obstruction is usually surgical. The doctor never gives a purge, for this will only make matters worse. If the trouble is an external hernia, the bowel may perhaps be pushed back, but otherwise the trouble can be treated only surgically. If no surgeon is at hand the patient is kept flat on his back, with the knees flexed, and an ice-bag is put over the lump. The position and the cold may relax the opening and allow the bowel to drop back. If we let the patient alone, sometimes the trouble will right itself, but so rarely that friends should never take the risk of its occurring.

ENTEROPTOSIS.—By enteroptosis is meant literally that the intestines have fallen, but the word usually means that the stomach, intestines, liver, kidneys, and all the viscera have, for some reason or other, taken too low a position in the abdomen. This occurs in two conditions. In the first group are women who are the mothers of several children, and cases of heart and liver trouble in which the abdomen has been considerably distended with fluid. Enteroptosis in these cases may be extreme, but, strange to say, the patient complains of no symptoms whatever. But enteroptosis occurs also (the second group) in young nervous persons, especially women, and they complain of various severe symptoms, such as dyspepsia, dragging pain, throbbing of the aorta, etc. In these cases the actual displacement of organs is less than in the first group. The treatment is to treat the neurosis and by proper binders to support the organs.

MUCOUS COLITIS.—Mucous colitis is almost always a symptom of neurasthenia, although very rarely it is due to some local trouble in the pelvis. Patients with this trouble complain that they pass slime with their stools, that sometimes the whole stool is nothing but mucus, although as a rule it is mixed with fecal matter. Sometimes the mucus is in long strings, sometimes in long tubes, just as it has peeled itself off the wall of the bowel. These patients are sure that they have passed a tapeworm, or that a portion of the bowel itself has been passed. They have usually severe colicky pains while the long sheets of mucus are peeling off. The patients usually have also nervous diarrhoea and other symptoms of neurasthenia. Their suffering is often extreme, and they are frequently operated on for appendicitis, gall-stones, pelvic trouble, or intestinal adhesions. We not infrequently see patients who have had all these operations performed.

INFARCTION OF THE BOWEL.—By infarction, as described on page 42, we mean that the blood-vessel supplying an organ—in this case the bowel—has for some reason become plugged, and hence the bowel wall in the area supplied by this artery receives no food. Unless operation is performed and the dead portion of the bowel removed, fatal peritonitis may result.

The symptoms are similar to those of obstruction, except that the patient passes more blood in the stools. It occurs

most often in heart disease and in any other condition which favors embolism. This condition is much more frequent in horses ("colic") than it is in men, but in the case of the former it is due to parasites which live in the blood-vessels of the intestine.

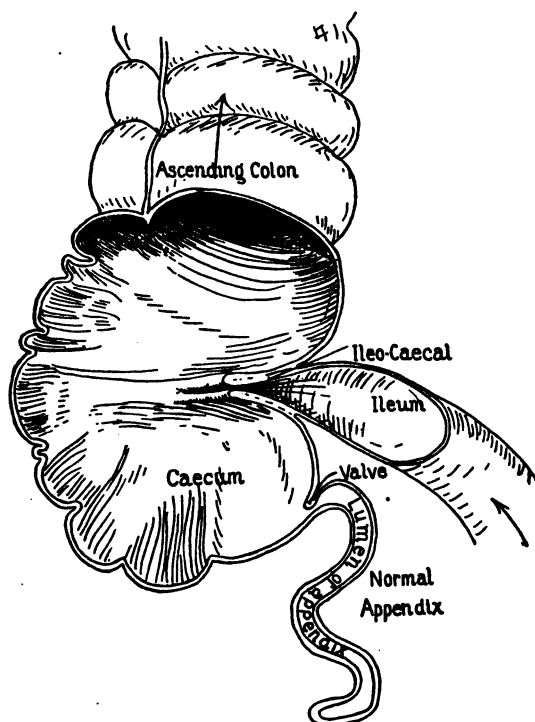


FIG. 61. The normal caecum showing the ileo-caecal valve and the appendix vermiformis.

APPENDICITIS.—The appendix (Fig. 61), is a small piece of bowel about four inches long and as large around as the little finger. One end of it is closed, the other opens into the large bowel just below the ileo-caecal valve. It lies free in the abdominal cavity. No function for it can be assigned in the case of man, but in certain lower animals it seems to serve as a pouch which increases the digestive surface of the bowel.

Chronic inflammation of the appendix (*chronic appendicitis*) is not at all uncommon. Its wall gets thicker and thicker,

and its lumen narrower and narrower, until finally the whole appendix is nothing but a thick, solid cord of scar tissue. This condition is known as "chronic obliterative appendicitis" (Fig. 62). Strange to say, the symptoms of this condition are not in the appendix or anywhere near it. Often they are gas-

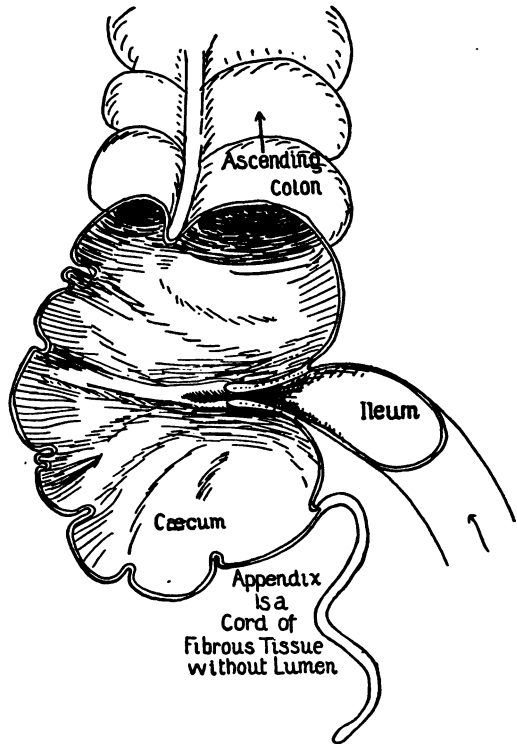


FIG. 62. Chronic obliterative appendicitis. As a result of repeated slight inflammations the appendix is a solid cord of scar tissue.

tric, and the patients complain of hyperacidity, of heart-burn, of dyspepsia; and many have repeated attacks of vomiting. All of these symptoms are probably due to reflexes from the appendix. Other patients—often the above also—have severe constipation which will not yield to ordinary treatment, and sometimes complain of extreme abdominal pain.

Acute appendicitis (Fig. 63) is quite a different matter, although it is most apt to occur in an appendix already the

seat of a chronic trouble. Acute appendicitis is caused by some germ which settles in the appendix and there sets up an inflammation with pus formation. If the appendix can be removed before the inflammation has gone through its

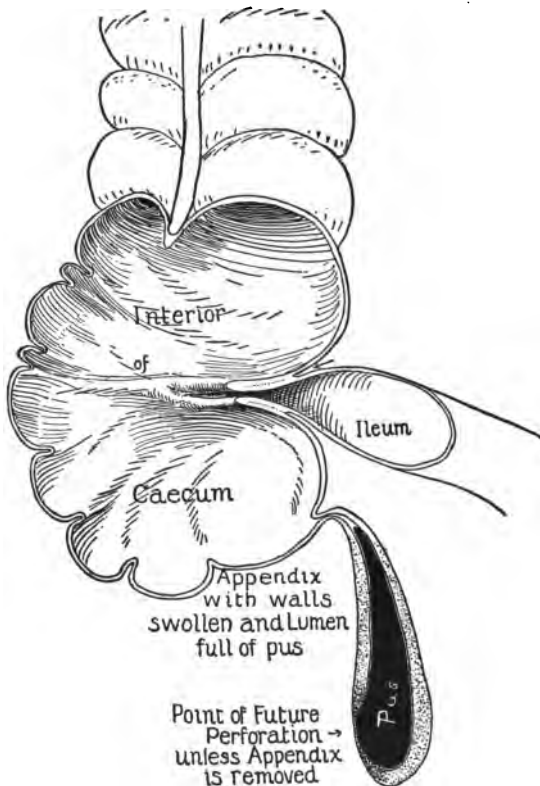


FIG. 63. Acute appendicitis. If operation is performed now the wound can be "closed" and in about a week the patient will be up and about. But if the operation is not performed, the appendix in a few hours will rupture and local (leading to abscess) or general peritonitis will follow.

wall, then there is no further trouble; but if we use medicinal treatment in the hope that it will subside, as it does in the majority of cases, there is then a good chance that the inflammation will spread through the wall and will affect the surrounding bowel, causing peritonitis or appendix abscess (Fig. 64). In the former case the abdomen can be closed as soon as

the appendix is removed, and the patient be out of the hospital in ten days; but in the latter case large amounts of packing must be put into the wound to allow the escape of the exudate, and the patient will have to remain in bed for six weeks. It

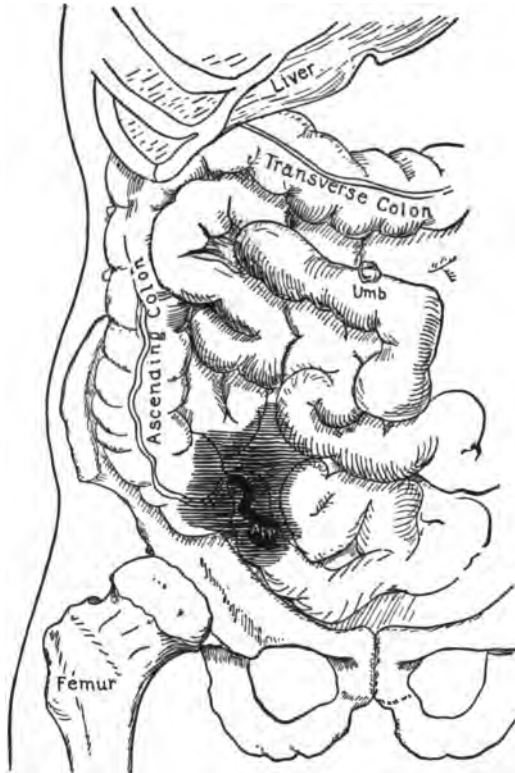


FIG. 64. Acute appendicitis, gangrene of the appendix, perforation, and spreading peritonitis. In this case the appendix was not removed, and the inflammation has spread to the surrounding loops of bowel. An abscess will form in the shaded area or the inflammation may spread through the entire peritoneal cavity.

is especially for this reason that operation is advised even in a very early mild case.

If the germ is very virulent, the disease may spread rapidly, the appendix fill with pus and become perforated, and the pus pour into the peritoneal cavity, setting up a general peritonitis even within twenty-four hours. If the process is not so very rapid, the loops of bowel around this appendix

will, because of a slow peritonitis, become stuck together—"adherent"—and form a wall against the spread of the trouble. Within this enclosure a large abscess will now form around the appendix, which contains the pus and also perhaps some intestinal contents. The trouble is now a local peritonitis, or "appendix abscess." These abscesses may contain even a quart of bad-smelling pus. But when the organism is very virulent, and the inflammation spreads so rapidly that the trouble cannot become walled off, then a general peritonitis results. We cannot know until we operate what organism is present, and, since there is always danger that it may be the virulent streptococcus, rather than the less virulent staphylococcus or the colon bacillus, it is better to operate at once, since the danger of the early operation is much less than that of the later.

The treatment in appendicitis is always operation. The only patient for which we should recommend medical treatment is one living where there is within call no surgeon whom we should trust. If that is the case, the patient should remain absolutely quiet in bed and receive no nourishment by the mouth. Under no circumstances should he take a purge, for this would make the trouble all the worse and greatly hurry the disease. If one cannot operate, the patient is made quiet and comfortable with morphia; but in case an operation is in question morphia is never given, no matter how much the patient suffers, because we need the mind perfectly clear to judge of the symptoms until the question of operation is decided upon; then it is given to relieve the pain.

People very often ask why we have so much appendicitis now, while a few years ago it was unheard-of, and the answer is that there is no more now than there was then. Formerly we heard of many who died of "inflammation of the bowels," of "general peritonitis," and of "abscess of the intestines"—conditions which are much more rare now that appendicitis cases are not allowed to run long enough to produce these fatal conditions. When a case begins we have no means of telling whether it will be merely a "stomach ache" and subside without further trouble, or whether it is one attack of a chronic trouble; whether there is no hurry, or whether within twelve hours there may be a general fatal peritonitis. Since there is no way of deciding early operation is always much the safest course.

The symptoms of acute appendicitis are often so slight that judgment can be passed upon them only by a competent surgeon. It is for that reason that we do not here give a list of the symptoms. It is enough to say that in severe cases there are nausea and vomiting, and usually constipation followed by diarrhœa; that there is usually pain on defecation and micturition; that there are pain and tenderness for the first few hours over the whole abdomen, often around the umbilicus or higher up or on the *left* side, and that later this pain and tenderness become localized on the right side.

CHAPTER VI

DISEASES OF THE LIVER, GALL-DUCTS AND GALL-BLADDER

The Liver is a large organ, which weighs about 1500 Gm., or three pounds. It is for the most part tucked away behind the ribs in the upper right-hand part of the abdomen.

The liver is made up of a myriad of small livers called lobules (Fig. 65), each just about large enough to see with the naked eye, practically all of them similar in size, shape, and function. Study one of these little livers, and we study the whole organ. Each lobule has somewhat the shape of a thimble (Fig. 66). It is composed of liver cells and vessels. Through its centre runs a tiny tributary to the hepatic vein, *a*. Along the outside run three or four tiny branches of the portal vein, *b*. The portal and hepatic veins are connected by a multitude of small capillaries, *c*, and around these capillaries the liver cells are arranged. We have, therefore (Fig. 67, *d*), liver cells, on one or several sides of which is a capillary, *c*. Through these capillaries flows practically all the blood from the stomach, bowels, pancreas, and spleen, and so every particle of food which is absorbed by the blood has to pass by these cells. From these liver cells start tiny bile ducts, the tributaries of the gall-duct.

The functions of the liver are various. Among these its glycogen-storing function is very important. We often eat at once large amounts of carbohydrates, practically all of which is transformed into glucose and collected in the blood of the portal veins. Were it not for the liver this glucose would get into the blood of the general circulation, which

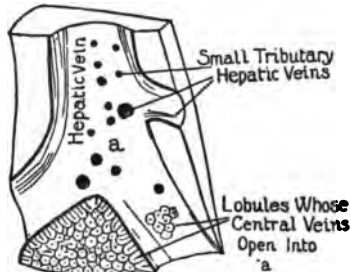


FIG. 65. A small fragment of liver very slightly magnified. The cut was made along a small hepatic vein. In the lower portion the lobules are represented.

then would at times be far too rich in sugar and at other times too poor. The liver removes from the blood practically all of the glucose which the blood has collected from the intestine, changes it into animal starch, or "glycogen," stores this up, and then doles it out again as glucose as occasion requires. The liver often contains about one-third of a pound of

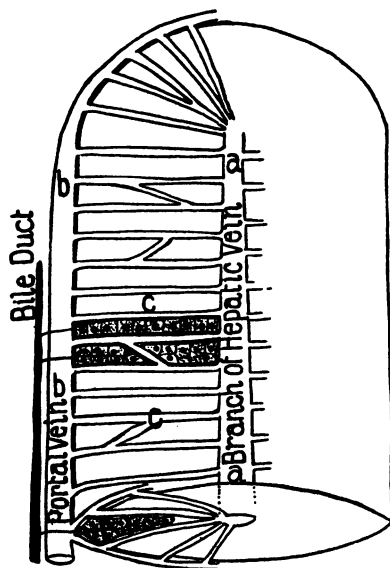


FIG. 66. One liver lobule, in vertical section. The blood flows from *b*, a branch of the portal vein, through the capillaries, *c*, to *a*, a branch of the hepatic vein. While in the capillaries it comes in contact with the liver cells. From these cells start the bile capillaries which flow to the bile duct.

glycogen—rather a large amount, considering that the blood of the entire body does not contain at one time over one-third of one ounce. When there is more glycogen than this to store up, the surplus is transformed into fat and stored up in other depots of the body. Glucose is the coal of the body, and the circulating blood must always contain a small amount, but not over 0.2 per cent. Any surplus over this the kidneys remove from the blood and excrete.

Again, all the food of the body and all of the living protoplasm itself are sooner or later burned up, and their ashes excreted through the kidneys. What all these ashes are is not known, but some, especially ammonia bodies, we know to be poisonous. If they were allowed to remain in the blood until the kidneys could get rid of them all, the body would suffer. It is one of the liver's functions temporarily to remove these ashes from the blood, to transform them into something—chiefly into urea—which is not poisonous, and to return this to the blood. This the kidneys then eliminate from the body.

A third function of the liver is to remove from the blood certain ashes and to excrete them in the bile. The ashes of

used-up hæmoglobin is one illustration. The liver cells split off and save the iron of the hæmoglobin, and the rest of the hæmoglobin is excreted as the green or yellow coloring matter of bile. Cholesterin and many other ashes are also eliminated in the bile.

Further, the bile is in a certain sense a digestive fluid. The bile acids which give vomitus its bitter taste probably are not excreta, but are manufactured by the liver to aid in the absorption of fats and soaps, for these, though not very soluble in water, are soluble in the bile. This must greatly aid the mucosa, for when a gall-stone prevents bile from entering the intestine only about one-fifth of the fat is used, and the rest is in the stools. The bile acids also keep the cholesterin in solution, and this is important, because cholesterin, when it is no longer in solution, forms gall-stones.

We thus see that the liver has two secretions: an internal secretion of glucose, urea, and perhaps many other bodies, which it turns back into the blood; and an external secretion, the bile, which flows down into the intestine. The external secretion, the bile, contains two groups of bodies; the secreta, or substances manufactured for a specific purpose, such as the bile acids; and excreta, or substances which are merely got rid of because they are no longer of any use. About 500 to 800 c.c. of bile are secreted every twenty-four hours.

From popular talk one might infer that liver trouble was rather common. One hears so often the word "bilious," and of remedies to "stir up the liver;" but the symptoms of which "bilious" persons complain are almost invariably those of constipation. There are many diseases of the liver, with severe symptoms, but, strange to say, symptoms which do not always suggest the liver as their source.

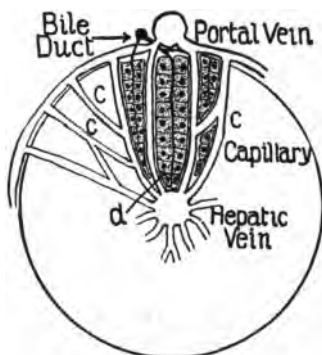


FIG. 67. One liver lobule in cross section. The blood flows from the branch of the portal vein through the capillaries, c, to the branch of the hepatic vein. While in the capillaries it comes in contact with the liver cells, d. In these cells arise the tiny bile capillaries which empty into the bile duct.

JAUNDICE.—Jaundice is a common symptom of liver trouble, but more often of troubles in the gall-ducts. The little bile ducts start in the liver cells, then unite, forming larger ducts, which continue to unite until we have the large hepatic duct, a tube about two inches in length and a quarter of an inch in diameter. Underneath the liver is the gall-bladder (Fig. 52), a pouch holding about 30 to 50 c.c. of bile, with a duct, the cystic duct, which is about half an inch long, and an eighth of an inch in diameter. The hepatic duct and the cystic duct unite together to form the common duct, a tube about three inches long and about a quarter of an inch in diameter. This duct opens into the intestine. The liver is practically always forming bile, but between meals much of this bile is stored up in the gall-bladder. After meals the bladder empties, forcing the bile down through the common duct into the intestine.

When the bile cannot flow into the intestine, but is dammed back into the liver, it is reabsorbed into the blood, is carried over the whole body, and tinges skin, the whites of the eyes, the tears, the saliva, the urine, to a yellow or a greenish yellow. This condition is called "jaundice." It has been customary to speak of two forms of jaundice, the hepatogenous and the hematogenous. Hepatogenous jaundice is due to obstruction in the large gall-ducts. This may be caused by a plug of mucus or a stone in the common or hepatic ducts, or by a tumor pressing against these ducts and closing them. A gall-stone plugging the cystic duct will of course not cause jaundice, because that will not dam the bile back into the liver.

In hematogenous jaundice the trouble is also an obstruction to bile flow, but in this case the obstruction is in the finest gall-ducts. Perhaps there is so much solid matter to be excreted that the bile is too viscid to flow and therefore plugs up the smaller ducts and dams the flow back into the liver cells and hence into the blood. Hematogenous jaundice is seen particularly after a great destruction of red blood-cells, such as occurs in severe fevers, malaria especially, and in any severe toxic condition, or of liver cells, such as occurs in acute yellow atrophy.

The common symptom of jaundice is the color, which when very slight is best recognized in the whites of the eyes.

If there is total obstruction of the bile flow, the skin may be almost any shade of yellow, green, or even brown. The stools will be white or clay-colored, for they normally owe their color chiefly to the bile pigment. The bile is poisonous and affects the heart, so that the pulse is slow. The blood also coagulates slowly, the skin bruises easily, and hence these patients bleed profusely. Surgeons refuse to perform any operation on such patients until by giving calcium lactate they have increased the coagulability of the blood.

Catarrhal jaundice is a jaundice which occurs commonly in young persons and lasts from four to eight weeks. There are very few symptoms, although there may at first be slight fever, and there are almost always nausea and a loss of appetite. As a rule, the original trouble is a slight inflammation of the mucous membrane of the stomach—an inflammation which extends into the duodenum. The orifice of the bile duct is a tiny hole in the mucosa, and this is easily stopped up by a little swelling of the mucous membrane or especially by a plug of mucus.

The treatment is simple. The patient should be kept quiet and the gastro-enteritis treated by proper feeding.

ACUTE CHOLECYSTITIS.—By this is meant an inflammation of the gall-bladder caused by bacteria which lodge in this pouch. The bile seems to be one of the chief exits of germs in the blood. Acute cholecystitis is very common in acute fevers, particularly typhoid, and especially if gall-stones are already present. It is a rather serious condition, for the bladder is apt to rupture and allow the pus to pour into the peritoneal cavity, causing general peritonitis. The treatment is surgical; the gall-bladder is opened, and the pus liberated.

The gall-bladder also is a very suitable home for germs. They can stay here for months or years without causing any inflammation of the gall-bladder. After an attack of typhoid fever, the typhoid bacilli can live in the gall-bladder for perhaps forty years. The stools of these persons contain immense numbers of these germs. This is a serious matter, for the persons are themselves perfectly well, and yet they can through years spread the disease broadcast, and are directly responsible for the outbreaks of this fever.

GALL-STONES.—Gall-stones are made up chiefly of cholesterolin and bile pigment. They vary in size from that of a

grain of sand to that of a pigeon's egg or even a hen's egg. Some are so soft that they can be crushed in the fingers, others are "hard as a rock." They form chiefly in the gall-bladder, but also in the larger gall-ducts. The gall-bladder may contain only one, but as a rule it contains a dozen or even two or three hundred stones. Gall-stones occur especially in those who have had typhoid fever, and in women who are the

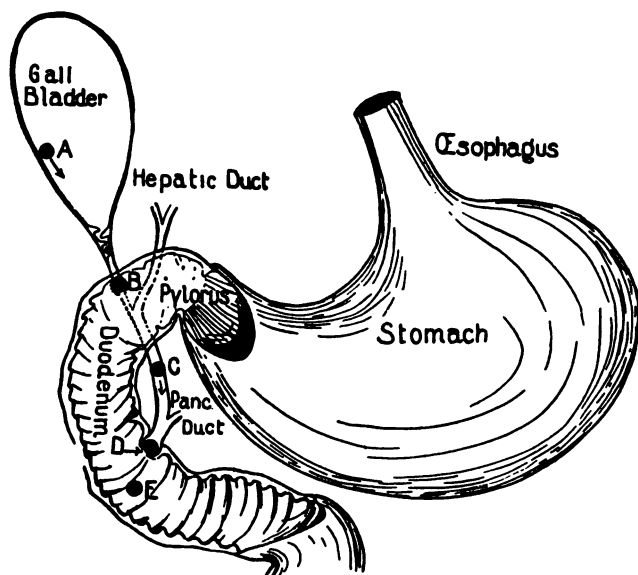


FIG. 68. Outline of the stomach, duodenum and bile ducts showing the various possible positions of gall-stones. *A*, gall-stone in the gall-bladder, which will cause no symptoms. *B*, stone in the cystic duct which will cause colic but no jaundice. *C*, stone in the common duct. This will cause less pain than *B*, since the duct is larger, but will cause jaundice. *D*, stone in the ampulla of Vater. This will cause "hepatic intermittent fever" and jaundice, and also will disturb the pancreatic functions. *E* the stone has reached the bowel. It will cause no symptoms.

mothers of several children. Germs, and the stagnation of bile seem the predisposing factors. The great wonder is that gall-stones are not commoner, for cholesterin is extremely difficult to keep in solution. Given a nucleus of bacteria or of little flakes of mucus, and around this will crystallize the cholesterin and bile pigment.

The symptoms of gall-stones vary greatly (Fig. 68). In the first place, a person may have the gall-bladder full of them and never know it if the stones stay there, unless their pres-

ence encourages an acute cholecystitis. They always cause a slow, chronic thickening of the gall-bladder, and this seems to be nature's way of handling them; for the walls of the gall-bladder get thicker and thicker, and contract down on the stones, holding them tight, so that they cannot wander into the gall-ducts. But as a rule one or more of these stones are carried by the current of bile into the cystic duct, then through the common duct into the bowel. If the stone is tiny the patient may not know of it, but if the stone is large enough the patient has "gall-stone colic." All of a sudden, like "lightning from a blue sky," is felt an extremely sharp, severe pain, running from the right side under the ribs to the right shoulder-blade. The patient sometimes rolls around on the floor, vomiting, and screaming with excruciating pain. The pain is probably caused by the stone as it squeezes its way through the small cystic duct. When once it has reached the common duct, it is more apt to travel along easily, for the common duct is about three times as big as the cystic duct. At the mouth of the common duct the stone reaches the ampulla of Vater (Figs. 68 and 73). This is a pouch in the mucous membrane of the duodenum, into which open the common bile and the pancreatic ducts. It opens into the bowel through a tiny hole. The stone can travel easily through the common duct, but it will have difficulty in forcing its way through that tiny hole into the bowel. When it has done so, the pain of course ceases at once. The patient is sure that it was the last treatment he tried that stopped the pain. In other cases the pain is not severe, but merely a dull ache. With gall-stone colic there are very often fever and vomiting, due to an infection started in the gall-bladder by the irritation which the stone sets up.

But the stone may reach the ampulla of Vater and stay there for months or years. We have then a symptom complex, "stone in the common duct," which is easily recognized. The ampulla is roomy and holds the stone and also allows the bile to flow fairly well around it, into the bowel; but occasionally, perhaps due to a slight swelling of the mucous membrane, the stone entirely plugs the orifice. The patient is slightly jaundiced all the time, but now has sudden, acute pains in the upper abdomen, the jaundice gets much deeper, the stools are clay-colored, the fever rises rapidly with a chill and sweat.

After a few hours the pain rapidly subsides, bile again flows into the intestine, and the jaundice somewhat clears up. This condition is often mistaken for malaria. It is known as the "intermittent hepatic fever of Charcot."

Some large stones are not able to force their way through the gall-ducts. They set up in the gall-bladder an inflammation which extends through to a loop of bowel or to the abdominal wall; then ulceration follows, and the stone escapes into the bowel; or is discharged through the skin.

The treatment of ordinary gall-stone colic is to make the patient as comfortable as possible with a hot bath and either

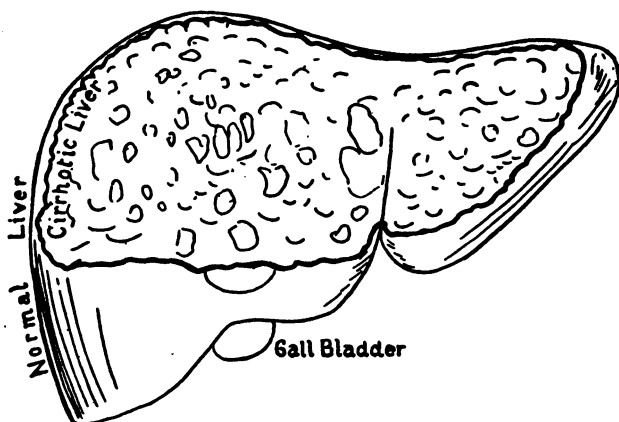


FIG. 69. The outline of a normal liver and of a cirrhotic or "hob-nail" liver to show their relative sizes, and differently appearing surfaces. (Much reduced.)

chloroform or morphia. A person subject to gall-stone attacks should between attacks avoid starches and sugars. The surgery of the gall-bladder is now so perfect that one can safely recommend that the gall-stones be removed by operation, and a recurrence of the trouble be thus prevented. If the stone is in the common duct, an operation is very necessary, although this operation is much more severe than one on the gall-bladder.

The diseases just mentioned are really diseases of the gall-bladder and gall-ducts, not of the liver.

ATROPHIC CIRRHOSIS OF THE LIVER is an important disease. It is due to various poisons—particularly, alcohol—which injure the liver cells. They gradually diminish in size, and

die. Their place is taken by scar tissue, and scar tissue always contracts. In time we may have more scar tissue than liver cells in the liver. The result is at first a large liver, then a small one which may weigh only from one to one and a half pounds. It is very hard, for it now consists chiefly of scar tissue. Also its surface is very rough, because the scar tissue within it is in rather coarse bundles which by contracting pull in the capsule in certain points and cause the remaining

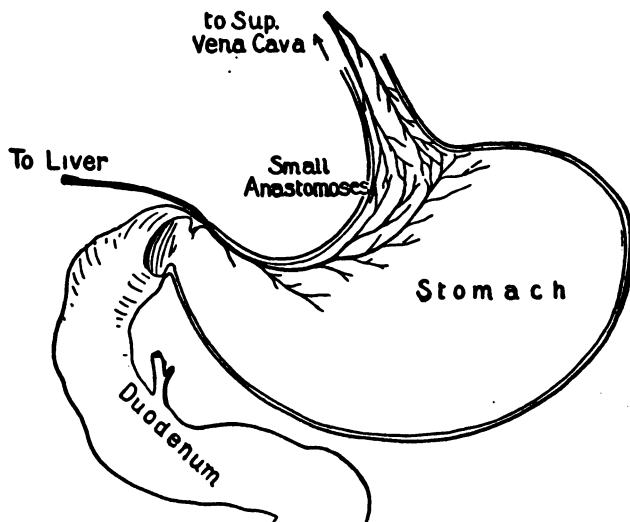


FIG. 70. Diagram of the stomach showing the venous circulation at the lesser curvature. It will be seen that the blood in the veins of the cesophagus flows upwards and empties into the superior vena cava without flowing through the liver, and that the blood in the veins of the lesser curvature of the stomach flows towards the pylorus and then through the liver. These two sets of veins are united by fine anastomoses.

liver tissue to bulge out in little lumps, hence the name "hob-nail liver" (Fig. 69).

The symptoms do not result from the diminishing amount of liver tissue, for a small amount of that is enough to perform the sugar and urea functions. The trouble is with the portal circulation. Practically all the blood from the digestive organs is collected in the portal veins and carried to the liver. Since these cirrhotic livers do not allow it free passage it is dammed back into the digestive organs, with the result that they are choked up with the blood and hence cannot functionate well. They become the seat of chronic passive congestion (see

page 61). The patient has gastritis, enteritis, diarrhoea; the spleen becomes swollen; a chronic peritonitis develops; fluid gathers in the peritoneal cavity (ascites), which may need to be tapped repeatedly; anywhere from five to twenty litres may be removed at each tapping. There will be also slight obstruction to the bile flow, and the person will be slightly jaundiced. But this is not all. At certain points in the abdomen the portal circulation does anastomose with

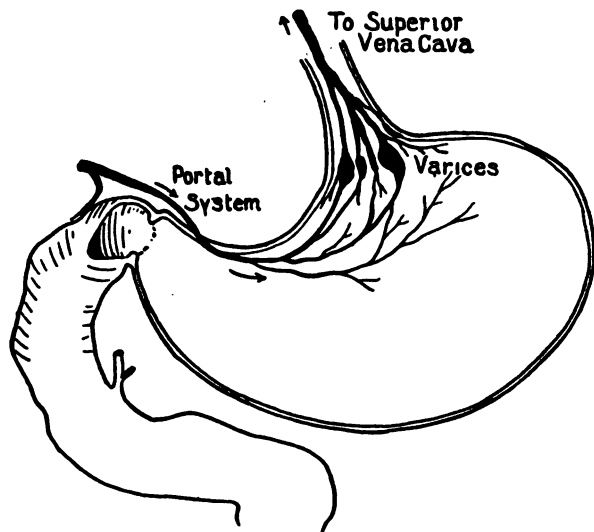


FIG. 71. Diagram of the stomach showing the venous circulation of the lesser curvature in a severe case of cirrhosis of the liver. The current of blood will now be towards the œsophagus, and the fine anastomoses will be distended into varices. These have thin walls and rupture causing profuse hemorrhages into the stomach.

the general circulation, and some portal blood can flow to the heart without passing through the liver. One of these points is the cardiac orifice of the stomach see (Fig. 70). The blood from the œsophagus flows directly to the heart, that from the stomach to the liver. Veins always anastomose, so that blood can choose between the two paths. If the portal circulation is dammed back, a great deal of the blood in the stomach wall will escape through the œsophageal veins; but these veins become distended, forming œsophageal varices (Fig. 71), whose thin walls very often burst. Hence patients with cirrhosis of the liver have terrible, often fatal

hemorrhages from the stomach. Also in the lower rectum is a point where portal and general circulation meet, and in cirrhosis of the liver venous varices form here also, which are known as hemorrhoids or piles, and these too may rupture and cause severe hemorrhage. Of course hemorrhoids are very common and in the majority of cases are due to simple constipation, but in cirrhosis of the liver they are constantly present. Another point where the blood has the choice of these two courses is in the navel. There are veins which run from the portal vein through the liver to the abdominal wall at the umbilicus, and in case of portal obstruction a large amount of blood escapes the portal route here. The result is a skein of veins which radiate from the umbilicus through the skin forming the "caput Medusæ." The blood in the vessels at the base of the mesentery can escape the hepatic route by flowing into the veins of the posterior abdominal wall.

Through all these anastomoses a compensatory circulation is established, through which the blood can get to the heart without passing through the liver. The surgeons try to imitate this process—try to open up new channels from the capsule of the liver to the abdominal wall and omentum—by scraping these surfaces so that adhesions will form. The adhesions will contain fine blood-vessels. The results are not yet satisfactory.

The chief symptoms of cirrhosis of the liver are, therefore, those due to obstruction of the portal circulation. In severe cases, however, toxic symptoms are present; that is, the patient becomes delirious and then comatose, and may die in convulsions. We suppose that these poisonous symptoms are due to the lack of liver function, but closer than that we cannot yet go. The outlook is bad in all cirrhosis cases unless the compensatory circulation is sufficient to relieve the congestion. It is especially bad if ascites has developed, although there are patients who have been tapped, first frequently, and then less often, until finally a compensatory circulation sets up, and for the rest of their lives they are practically well. There is always danger that one of these much distended veins may rupture.

THE HYPERTROPHIC CIRRHOSIS OF HANOT.—This is a very rare condition, and quite a different disease from the above. The liver is very much larger than normal, in fact it may weigh

from 2000–4000 Gm. It contains an enormous amount of new scar tissue, but this, instead of being in bands as in the atrophic form, is quite uniformly scattered throughout the whole organ, and hence the liver is large, smooth, and very firm indeed. There are much less obstruction to portal circulation and much worse toxic symptoms than in the atrophic form. No cause can be assigned; we know it is not due to alcohol. It is a very chronic disease, lasting from four to ten years. The jaundice is slight; there is some pain in the liver; there are some nausea and vomiting, and a rather marked tendency to hemorrhage; there is no ascites, but usually a terminal fatal cachexia with intense jaundice.

ABSCCESS OF THE LIVER.—The liver is a great sieve through which flows all the blood from the digestive organs. The intestine is always full of bacteria, most of them harmless, but some virulent, and all waiting for a chance to gain a foothold and make trouble. Since large numbers are occasionally, perhaps frequently, picked up in the portal blood, it is no wonder that the liver is so often the seat of infection. The great majority of these bacteria are doubtless killed at once by the blood-plasma, but some get a foothold in the liver and multiply there. Whenever an ulcer forms in the stomach or intestine the germs can get into the blood stream. If an abscess develops anywhere along the gastro-intestinal tract there will quite certainly soon be others, usually larger, in the liver. And more often the “portal of entry” cannot be found. The toxins of the germs kill the liver cells in their neighborhood, and this dead tissue seems a protective wall for them. Meanwhile leucocytes have been migrating from the blood-vessels into the infected area to aid in the protection of the body. The result is soon an abscess cavity full of a liquid consisting of living and dead pus-cells, of liquefied liver cells, and bacteria. Open this cavity, and this liquid “pus” will flow out, leaving an empty hole.

When a gall-stone gets caught in a gall-duct, it causes at once an inflammation, which may extend into the smaller gall-ducts. Each duct may be filled with pus, that is, it is really an “abscess.” This condition is called “cholangitis.” Sometimes (and especially in these gall-stone cases) these abscesses are really in the portal vein themselves, a condition known as “pylephlebitis.”

These abscesses may be due to several kinds of germs, but the most common cause is not a bacterium, but an animal, *Amoeba coli*, the most important cause of dysentery. Sometimes this animal causes multiple abscesses, but more commonly only one, often a large one, even as large as a man's head.

The symptoms of liver abscess will be general and local. A great deal of poison forming in the liver is carried around the body in the blood, and gives rise to the symptoms of fever. The skin soon becomes pale and jaundiced. The temperature is daily very high and then very low, and there are severe chills and sweats, which simulate those of malaria. A large abscess may remain undiscovered a long time. While the abscesses are small, nothing may suggest that the liver is the seat of disease; but soon this organ swells and becomes painful. Where the inflammation reaches the surface there will be some pleurisy or peritonitis. The body handles the problem by forming a dense wall of scar tissue around the abscess. In this way its advance is often checked, the temperature no longer rises, the pain ceases, the patient feels well and considers himself well; but the abscess is still there and may at any time cause trouble. As a rule, however, the abscess is not checked, but extends in some one direction and finds an outlet—"comes to a head"—and it is a matter of vital importance where the "head" is. Fortunately, the abscess often extends upwards. The zone of inflammation, that is, the zone where the disease is extending and the liver is fighting, always extends ahead of the actual abscess and soon reaches the upper, outer surface of the liver. This rubs against the diaphragm, which also becomes inflamed. Since two inflamed surfaces which touch each other are soon stuck tightly together, so liver and diaphragm are now "one piece." Then the inflammation extends through the diaphragm, and we have a pleurisy above it; but the lung pleura rubs against the diaphragmatic pleura, and so their surfaces stick tightly together. Soon liver, diaphragm, and lung are practically one solid piece. The abscess formation has meanwhile followed the inflammation, and a hole is soon eaten through into the lung. If the union of the liver, diaphragm, and lung is tight, the pus will not pour out into either peritoneal or pleural cavity, but will pour into a bronchial tube

(Fig. 72). In this way it finds a "vent," and the patient soon begins to cough up all this liquid pus. If successful he will empty the abscess, leaving a clean hole. The wall of scar tissue will now get stronger and contract, until some day

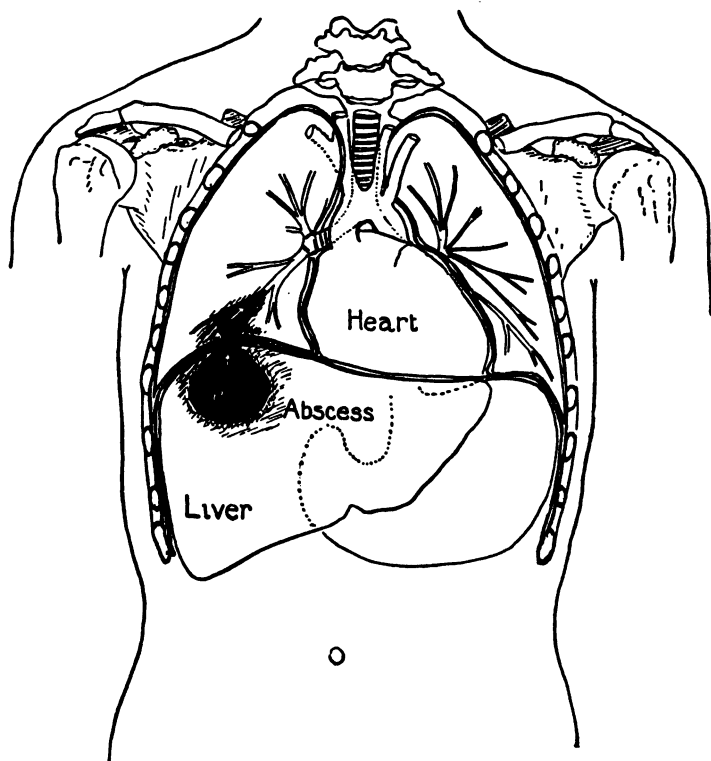


FIG. 72. Diagram of an abscess of the liver perforating through the lung into a bronchus. The preceding inflammation will have obliterated the peritoneal and pleural cavities in the path of the abscess.

the hole will all be gone, only a scar will remain and the patient will be well.

But the abscess may extend to a side of the liver or in a downward direction. Then the same process occurs. The liver becomes united by "adhesions" of inflammation to the stomach, to the intestine, or even to the wall of the abdomen; and then the abscess will perforate into one of these organs

or even make a hole through the abdominal wall and "come to a head" in the skin. Sometimes nature does not succeed so well, and the pus finds free vent into the pleural cavity, causing empyema; or into the abdominal cavity, causing peritonitis and death.

The treatment is operation. If one is quite sure there is a large pocket of pus in the liver, the surgeon should provide it with a hole through which it can escape freely and safely.

CANCER OF THE LIVER.—Very rarely do cancers originate in the liver, and then their starting-point is usually in the bile ducts. But secondary cancers in the liver are very common indeed. The primary growth may be almost anywhere, but the liver is pretty sure soon to filter from the blood or lymph channels some of the free cancer cells. The liver is an ideal place for these cancer metastases to grow. Often it is studded with these cancer nodules which grow to such large size that the liver, instead of weighing about three pounds, may weigh from ten to even forty pounds.

CHAPTER VII

DISEASES OF THE PANCREAS

The Pancreas, or the “stomach sweetbread” (Fig. 73), as it is called in animals, is situated behind the stomach. This gland has at least two functions: it manufactures the pancreatic juice, its external secretion; and it secretes a very im-

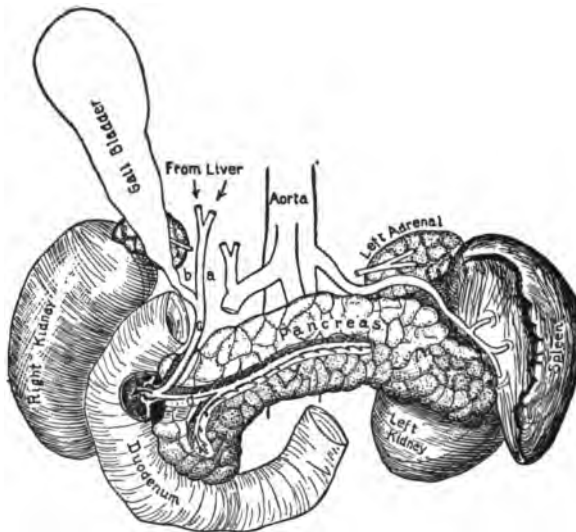


FIG. 73. Diagram of some of the abdominal organs. The stomach and the liver, which normally cover these organs, are not represented. *a*, hepatic duct; *b*, cystic duct; *c*, common duct. The arrow is in the ampulla of Vater. *d*, pancreatic duct, which is represented as exposed.

portant internal secretion which it pours into the blood stream. The pancreatic juice has already been described on page 111.

The internal secretion—a remarkable product—is entirely distinct from the external secretion. If the pancreas is entirely destroyed by disease, or entirely removed, the tissues of our body cannot burn grape sugar, their ordinary fuel. This accumulates in the blood, and the kidneys get rid of it by

excreting it in the urine. But if in the case of animals deprived of the pancreas a little piece of that organ is successfully grafted in any part of the body, under the skin, for instance, the internal secretion of this transplanted organ will save the animal from diabetes.

HEMORRHAGE INTO THE PANCREAS.—Occasionally persons who are apparently in perfect health die suddenly and without evident cause. Other apparently well persons complain suddenly of an agonizing pain in the upper abdomen, and this is followed by severe retching and vomiting. The nausea and vomiting may be terrific in severity. Death may follow within a day, or the patient may live two or three days in great agony, with all the symptoms of intestinal obstruction, including fecal vomiting. In the case of those who die suddenly the diagnosis of heart disease is usually made when no autopsy is performed, and in the great majority of cases this is correct; but in the case of a very few the pancreas is, at autopsy, found soaked in blood, which may also fill the abdominal cavity. In the case of those who live a few days or so, one finds, in addition to the hemorrhage, an active inflammation of the pancreas; and finds in the fat of the abdomen, of the abdominal walls, and even of the chest, little masses of "fatty necrosis." Part or even the whole of the pancreas may be dead. If the patient after the attack lives a little longer than a few days, an abscess is usually found in and around the pancreas, which may be floating, a mass of dead tissue, in the pus. All these conditions seem stages of the same process, one differing from the others according to its duration. The rapidly fatal cases are called "hemorrhage into the pancreas," or "apoplexy of the pancreas;" those which live a day or so have "acute hemorrhagic pancreatitis" (though in some cases there is no hemorrhage); the next stage is "necrosis of the pancreas;" the last, "abscess of the pancreas." Some get well, but the vast majority do not, unless saved by operation.

We know something of the cause of these cases. As is said on page 109, the trypsin of the pancreatic juice is the most powerful ferment known. This is formed as harmless trypsinogen, which cannot digest anything, and which in the bowel is "activated," or changed, to active trypsin. The change is made by a ferment supplied by the walls of the

bowels. The walls of the bowel are able to withstand trypsin; but suppose that by accident the trypsinogen is activated while in the pancreas. That organ cannot withstand its powerful action and is quickly digested in spots; the blood-vessels' walls are eroded, and hemorrhages follow. The trypsin diffuses freely in the body, digesting the fat in spots. Lipase, also a ferment of the pancreatic juice, spreads with the trypsin, and splits the fat of these digested masses to glycerin and soap, just as it is split in the intestines, when these act on it normally. The dead pancreas is now an excellent place for germs to grow in, so that inflammation and abscess are the result. Moreover, trypsin not only injures through its digestive action, but is also a powerful poison and is supposed to cause death by poisoning.

The treatment is to operate, slitting the pancreas open from end to end, and allowing all the fluid or pus to escape.

The reason why trypsin is thus prematurely activated is sometimes evident. Bile can activate this ferment, and has a chance to do this when a gall-stone plugs the common opening of both bile and pancreatic ducts, for then the superior force of the bile current forces bile back into the pancreatic duct. Bacteria, also the pus-cells which collect where there are bacteria, can activate trypsin.

PANCREATIC STONES.—The stones which form in the pancreatic duct are composed of calcium carbonate, or lime. The pancreatic juice is rich in this mineral, which in the intestine helps to neutralize the acid from the stomach, and which in fat digestion, helps to form soap.

Pancreatic stones can cause a good deal of pain in the upper abdomen, and this pain is usually ascribed to gall-stones. If a stone completely plugs the pancreatic duct, the pancreatic fluid is dammed back and the pancreas begins to atrophy—that is, to disappear. In a few months there may be none of this organ left.

Sometimes a cancer forms in the pancreas and destroys it; sometimes (usually as the result of injury) a large cyst—a sac full of fluid—forms, and this also may destroy the pancreas.

CHRONIC INTERSTITIAL PANCREATITIS.—This disease resembles cirrhosis of the liver in that the real pancreas tissue disappears, and its place is taken by scar tissue. In moderate grades the organ is large and hard, but in some cases all that

is left is a small mass of scar tissue. This may be the result of a chronic obstruction of the pancreatic duct, or of chronic mild infection by the germs which inhabit the gall-ducts in nearly all gall-stone cases. The result is a slow diminution of the secretion of the pancreatic juice, and later of the internal secretion of the pancreas.

When, because of disease of the pancreas, or obstruction to its duct, no pancreatic juice at all is furnished the intestine, for a long time there may be no symptoms. The gastric and intestinal juices can do most of the work of the pancreatic juice without the digestion suffering. The chief difference will be in fat digestion, for less than one-fifth will be used, and the rest will appear in the stools. But later the effect of the loss of the pancreatic juice becomes more apparent. The "diarrhœa pancreatica" begins. That is, the patient four or five times a day will pass stools which are huge in size, because so little of the meat and fats which are eaten is digested, and because they contain much gas, the result of fermentation. As so little food is digested, the patient will soon be very thin, no matter how much he eats, and will slowly starve to death. Later begins the "fatty diarrhœa." The fat, no longer split, is passed in a state like liquid oil or melted butter, either with the fecal matter or alone, and may continually ooze from the rectum. It quickly hardens as the stool cools.

If the pancreas is so completely destroyed that none of its internal secretion is formed, then the patient becomes diabetic. But for this result the destruction must be complete, for even a small amount of pancreas is enough to furnish sufficient internal secretion. Since in the majority of cases of true diabetes mellitus the pancreas, so far as one can tell by superficial inspection, is perfectly normal, surely the essential cause of diabetes is not total destruction of this organ. It is supposed that the internal secretion of the pancreas is not formed by the same tissue as the external, but by little areas scattered throughout its substance, called "islands," and that only when most of the islands are destroyed does diabetes begin. Some forms of pancreatitis seem to destroy these islands without injuring the rest of the pancreas. Some form of pancreatitis may explain most cases of diabetes, but that diabetes mellitus is always due to disease of the pancreas is yet to be proved.

CHAPTER VIII

THE KIDNEYS

THE WORD "FOODS" suggests to us the various meats, grains, vegetables, fruits, etc., which we eat. These are mixtures of the "food-stuffs," which are classified as proteins, carbohydrates, fats, salts, and water. A piece of meat, for instance, contains all these food-stuffs. The multitude of the proteins vary according to their origin, taste, appearance, etc., but chemically they are fairly similar, while biologically they are the living protoplasm of the animal or plant from which they come. There are very many different sugars in the various vegetables and fruits, and the oils and fats of no two vegetables or animals are exactly the same, but chemically all these food-stuffs can be classified under the five headings given above.

Another division of foods—a physiological division—would be foods which the body uses as fuel; and foods with which it repairs its used-up protoplasm. The former may be burned at once or stored up in the body for future use, but while stored and when burned they are only fuel, and never become any more a part of our living body than does the coal in the bin or in the fire-box become a part of the engine. The foods of the second group do become a part of our living protoplasm, just as new bolts, new pipes, new boiler plates, become a part of the engine. There is one difference, however, between our body and an engine; an engine never burns steel, while the body finally burns up for fuel all its worn-out protoplasm. Thus all food is, sooner or later, fuel. Some is used at once; some, the fat especially, is stored up as fuel and may not be used for years, and some is first made a part of our protoplasm, and then is burned. In the animal body only proteins can be used in the manufacture of protoplasm, which itself is a protein. Plants can manufacture protoplasm from the simplest substances—"bad air," water, and simple salts; this is their great duty

in the universe. Animals cannot do this or can do it only in slight degree. The herbivorous animals must steal it from the plants, while carnivorous animals in turn steal it second-hand from the herbivora, or third-hand from weaker carnivora. Such is the cycle of life; plants make, and animals appropriate. Of course during this metamorphosis the protoplasm does not remain alive, nor does it remain intact. The protein of broiled beefsteak or of boiled peas, for instance, is dead; our body tears these very complex, diverse proteins down into their constituent building materials (themselves pretty complex), and the cells of our intestinal wall build out of these fragments the few proteins peculiar to our bodies. These dead proteins are carried around the body in the blood to the cells of the various organs, which select what they need and incorporate it into their living structure. Fats and carbohydrates and probably albuminoids (gelatin et al.) are never more than fuel. Water and salts are practically unchanged while in the body.

Foods may be classified also as nitrogenous and non-nitrogenous, according to whether or not they contain nitrogen, one of the main elements in the proteid molecule. Fats and carbohydrates contain none. So important an element is nitrogen that it is used as a measure of protein. For instance, a beefsteak contains protein, carbohydrate, and fat. To find how much of it is protein we find out how much nitrogen it contains; this figure multiplied by nine gives the weight of the protein. Again, if we find in the total urine of one day fourteen grammes of nitrogen, we know that the body has during the past twenty-four hours burned 126 grammes of protein.

All of the food absorbed by the intestine is, sooner or later, burned, and in this combustion ashes are formed which must be removed from the body just as regularly and completely as must ashes be removed from a furnace. In the present chapter we are to study the organs which remove these ashes. But the ashes of the cells—the furnaces—are not the same as those found in the urine. The ashes of proteins are water, carbon-dioxide gas, and a number of very simple ammonia bodies of which we know little except that they are distinctly poisonous. Lest these injure us, the “ash heap” must be “worked over.” This is done by the liver, muscles, and

perhaps other organs (see page 130). They remove from the blood these poisonous ammonia ashes and build them over into the non-poisonous substances which we find in the urine. The ashes of protein when excreted are water, carbon-dioxide gas, urea, uric acid, creatinin, xanthin, hypoxanthin, adenin, hippuric acid, and a lot of other bodies—some known, others not yet isolated; also chlorides, sulphates, phosphates, and other mineral salts.

The ashes of fats are water and carbon dioxide. The ashes of carbohydrates are the same.

Water is already an ash (of hydrogen), and hence cannot be burned, but is taken, used, and is eliminated as water. The same can be said of the most of the mineral salts (*e.g.*, table salt, or sodium chloride).

The organs of excretion which free our body from these ashes are the kidneys, lungs, intestines, liver, and skin. The lungs excrete the most of the carbon-dioxide gas and about one-fifth of the water. The skin eliminates some of the water and a very small part of the salts. In Bright's disease it may get rid also of some of the nitrogenous waste, for near death sometimes urea crystallizes out as "urea-frost" on the skin, forming little solid masses about the size of the head of a pin, thickly clustered over the skin of the face. Since the skin can do some of the work of the kidneys, we always sweat the patient when the latter organs are diseased, to make it do even more.

The intestine also is an excretory organ. We do not refer now to the excrementa, the refuse which could not be digested or absorbed, and which merely passes through the intestine, but to some salts and ashes which the wall of the bowel removes from the blood. Of the salts, those of lime are, for the most part, excreted by the bowel wall. When the kidneys are in trouble the intestine also can do some of their work, and so the patient with Bright's disease is purged that the lower bowels may help still more. We know that the liver also is an excretory organ, for the bile contains a considerable amount of waste.

The kidneys get rid of the most of the ashes of the body, and this waste we call URINE. As urine are excreted about four-fifths of the water, a little of the carbon dioxide, and by far the most of the other ashes and salts—all but the little

the skin and bowels remove—and also nearly all the poisons and abnormal soluble bodies which by accident have got into the blood. All these bodies are in solution in the water of the urine.

UREA is a body which deserves special mention. Isolated in pure condition, it is in beautiful white crystals, which are very soluble in water. It is not an original ash, but a non-poisonous modification of the ammonia ashes. Urea is not at all poisonous, and yet the condition uræmia, a severe auto-intoxication, is named after it. Urea is composed of nitrogen, carbon, hydrogen, and oxygen, and about one-half of it by weight is nitrogen. Nearly ninety per cent. of the total nitrogen eliminated from the body is in urea, and since it is very much easier to find out how much urea is in the urine than how much nitrogen, for years doctors determined the amount of urea alone. A normal person voids between twenty and thirty grammes (almost an ounce) of urea every day, and every gramme represents three grammes of protein which have been burned. A person on poor diet may void but from fifteen to twenty grammes, one on very rich diet even one hundred grammes of urea a day. The amount of urea in the urine depends on two things; the amount there is in the blood to be excreted, and the ability of the kidneys to get rid of it. *Uric acid* can be crystallized from the urine in brown plates and needles, but when free from its coloring matter it is in white crystals. It is quite certainly an ash of the nuclei of cells. Not over one gramme is present in the urine each day. No substance in our bodies, perhaps, has been the subject of quite so much romance as this acid. Forty-six different diseases have been supposed to be due to it, and the list includes gout, rheumatism, nervous disorders, and even one eye trouble. It is known to be increased in gout, but so far as the other diseases go it may possibly be innocent of any importance. It is interesting that in reptiles—snakes, for instance—the major part of the nitrogen is eliminated as uric acid rather than as urea. *Creatinin* and *fifteen or twenty other nitrogenous substances* in the urine are interesting but not yet important. Of these there are perhaps in all five grammes eliminated each day.

Of THE SALTS sodium chloride is the most important, and of this the urine contains about fifteen grammes (one-half

ounce) each day; of sulphates it contains perhaps five grammes, of phosphates perhaps two, and of many other salts only traces—their number and amounts depending, of course, on the food we eat. *Sugar* is present in the urine in several conditions, the most important of which is diabetes mellitus. Normally the kidneys will allow scarcely a trace of glucose to pass through, but they are a safety-valve so far as glucose is concerned, and when there is more than 0.2 per cent. of this sugar in the blood the kidneys allow all the surplus to pass into the urine. It accumulates in the blood because it cannot be burned; it is excreted in the urine as a protection to the body. The kidneys may be perfectly normal in diabetes mellitus, although the extra work they have to do often injures them sooner or later. In various diseases of the nervous system, *e.g.*, after injuries to the head, there may for a time be a trace of sugar in the urine.

The normal renal cells will not allow more than the merest trace of ALBUMIN to pass through, and the trace which does pass cannot be detected by the ordinary tests. If there is even the merest trace recognizable by the ordinary tests, something is wrong. It may not be anything serious, and may be very temporary, but in the great majority of cases the cause of albuminuria is nephritis, or Bright's disease. On the other hand, the kidney may be the seat of a severe nephritis without any albumin appearing in the urine.

There are some persons who throughout their lives have albuminuria at times. In some cases the urine secreted while the person is in the erect posture contains albumin, that secreted while he is lying down contains no albumin. The cause of this so-called "orthostatic" albuminuria lies, perhaps, in the circulation of the kidney rather than in a disease of the kidney itself. Again, any one can produce a temporary albuminuria if he "overdoes" sufficiently. This is especially true of bicyclists, foot-racers, foot-ball players, soldiers on forced marches, after too long a swim in cold water, even after mental overexertion, and sometimes after overeating. The cause in such cases is temporary, and the albuminuria is slight and ephemeral. Some young persons have, for a long time, a slight albuminuria. In some this is due to an inherited renal trouble, in others to a nervous trouble, in others to circulatory troubles, and in still others to a floating

kidney. In many cases the condition later disappears, and the patient is well; others are never in the best of health.

All the above are called cases of "functional albuminuria," because the kidney is, so far as we can see, normal. In most cases the kidneys are not normal. In others there is heart trouble, and renal cells cannot act normally when all the capillaries supplying them with food and oxygen are overfilled with dammed-back blood. Many cases of fever of any sort have, during the fever, albumin in the urine. Here for a while the kidneys are not normal. More cases with slight albuminuria had during childhood scarlet fever, measles, diphtheria or tonsillitis, which perhaps caused a slight and permanent nephritis.

But most cases of albuminuria have true Bright's disease, or nephritis. The amount of albumin in the urine of these cases varies enormously. In the acute cases there is most, even so much that when the urine is boiled it truly solidifies. The more chronic cases have less, and the very chronic cases least—so little that only an expert can find it, while it may be absent for months at a time. One thing is certain; from the amount of albumin one can never judge the severity of the case. A case of acute nephritis which recovers may have maximal amounts; a case dying with severe chronic nephritis may have only a trace.

One hears a great deal about the casts in the urine. These bodies get their name from the fact that they are formed in the tubules as a mould. In certain abnormal conditions of the kidneys the renal tubules become at points filled with a substance which hardens, thus forming "casts" of the tube. The casts are then washed out, and can be found with a microscope in the urine.

In some cases many kidney cells become detached, and masses of them make up an epithelial cast (Fig. 74). The cells decay and become soon a mass of granules, and we have a granular cast, *b*, or the cells become very fatty, forming "fatty casts." The most common cast is of a pale, translucent material, perhaps a substance which has oozed from the blood into the tubule. These are "hyaline casts," *c*, and are hard to see. Some look as if made of wax, and so are very conspicuous objects, the "waxy casts," *d*. Blood casts and pus casts are terms requiring little explanation.

Casts have much the same clinical meaning as albumin, although either may occur without the other.

In the urine may be found RED BLOOD-CORPUSCLES,—either a few, as in acute inflammatory conditions; or many, as in cases of renal stone or of tuberculosis of the kidney; or very many, as in cancer of the kidney or bladder. There may be so many that the urine looks like blood. When the blood is sufficient in amount to change the color of the urine, the condition is called “hæmaturia.”

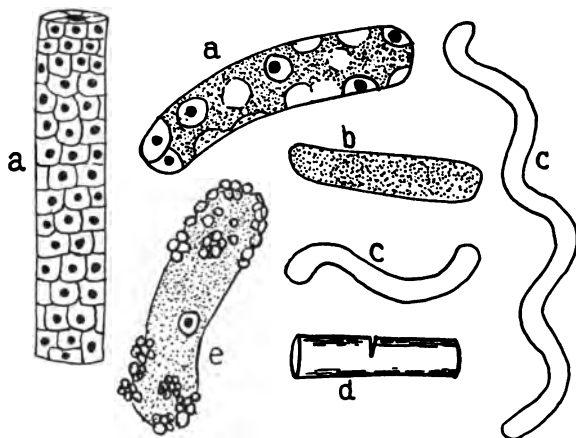


FIG. 74. Casts found in the urine. *a*, epithelial casts; *b*, granular cast; *c*, hyaline casts; *d*, waxy cast; *e*, fatty cast. (Much magnified.)

PUS-CELLS are always present in nephritis, as in other inflammatory conditions. When many in number, the condition is called “pyuria.” Pyuria occurs in pyelitis, in tuberculosis of the kidney, and especially in cystitis and urethritis.

EPITHELIAL CELLS also occur in the urine. Some are normal, for these cells are constantly shed from the surfaces of the ureters, bladder, and urethra, as from all epithelial surfaces. They are greatly multiplied in all inflammations.

The next question to answer is, HOW DOES THE KIDNEY DO ITS WORK? How does it separate from the great mass of blood almost all the waste and allow nothing of value to the body to escape? The kidneys, like other organs, are made up of little kidneys, and to understand one of these is to understand the whole kidney. Each of these little kidneys

is made up of living cells arranged in tubes. Fig. 75, *a*, represents the upper end of a tube. Its wall is made up of these renal epithelial cells, on the outer side of which is a fine network of capillaries, *b*, through which blood is constantly flowing. These little living cells are constantly bathed on their outer surface by the blood-plasma. They remove from this practically all the solid constituents of the urine, all of which are poured into the lumen of the tube and will flow to its open end. At the upper end this tube swells out

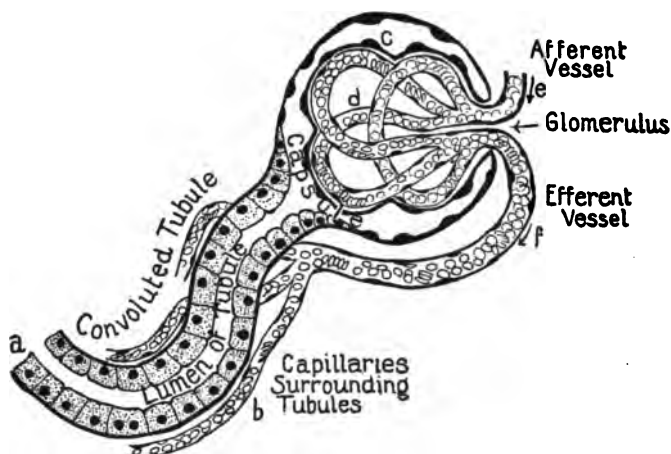


FIG. 75. Upper end of a convoluted tubule together with a glomerulus. *a*, convoluted tubule; *b*, the capillary surrounding a tubule; *c*, "Bowman's capsule"; *d*, the capillaries which make up the glomerulus; *e*, the afferent artery which breaks up into the capillaries *d*; *f*, the efferent artery which breaks up into the capillaries *b*. These capillaries *b*, unite to form the renal veins.

into a hollow ball called Bowman's capsule, *c*. At that pole of this ball which is opposite the point where the tube enters, the wall is pushed in, much as a child can push one-half of a rubber ball into the other half, by a knot of capillaries, "the glomerulus," *d*. These glomeruli can be just seen by the naked eye as red dots about the size of a pin prick. The wall of this hollow ball is lined with flat epithelial cells quite unlike those lining the tube. The capillaries of the glomerulus, all of which branched from one vessel, *e*—a branch of the renal artery—united again into one vessel, *f*, which leaves the capsule, flows down to the tube, and breaks up again into the network of capillaries, *b*, surrounding the tube. Through the

walls of the capillaries of the knot pours out into the capsule the most of the water and salts of the urine. This flows down into the tube and unites with the very concentrated solution of ashes there. This urine then flows down to the open end of the tube.

The capillaries surrounding the tubules then unite to form the renal vein, the blood of which is practically free from

waste. A great amount of blood flows through the kidneys, nearly ten times as much as through other organs of the same weight. In this way the kidney keep the entire blood pure. Just how their cells separate the waste products and allow nothing else to escape, is not known. But we do know that it is because they are alive; for the chemical and physical laws, as we now understand them, will not explain it. All secretory cells look much alike and yet they make no mistakes. They all have the same blood to deal with. The cells of the stomach separate out the materials for gastric juice; those of the pancreas, the constituents of pancreatic juice; those of the liver, bile; and the kidney cells take what the others reject and nothing else. The blood contains large amounts of albumin, some sugar, and many other substances, but the normal renal cell allows none of these to pass. These renal cells themselves require much food and oxygen. Pinch

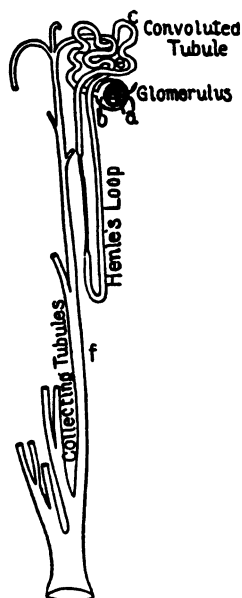


FIG. 76. The "unit" of the kidney. a, glomerulus; b, capsule; c, convoluted tubule; f, collecting tubule.

the renal artery for half a minute, and it is forty-five minutes before the renal cells start to work again; and when they do, the urine they secrete shows how injured they were.

In some very simple animals one such tube as we have described, or a row of them, is all the kidney the animal requires, but for man great numbers are necessary, and, to economize space, the tubes are not straight but twisted. Their course is much as follows. The tube which leaves the capsule (Fig. 77 b), is very tortuous and is called the "convoluted

tube," (Fig. 76, c); it then makes a long, straight loop, the loop of Henle, (Fig. 77, d); then again it is convoluted, e; then it opens into a long, straight tube, the collecting tube, f, which grows larger and larger in its course because it receives from all sides many convoluted tubes. An organ made up of a mass of these tubes all flowing in the same direction must of necessity be pyramidal in shape, since the convoluted tubes, which take up most room, and the glomeruli, are all in the outer layer. In the dog, for illustration (Fig. 78), the kidney is shaped like a pyramid or cone. In this we can distinguish the outer zone, the cortex, where are all the glomeruli, the convoluted tubules, and the conical end, the pyramid, b, where are the loops of Henle, and the converging collecting tubules which open in the surface of the pyramid. The tip of the pyramid projects into a dilated sac, the pelvis, c, which becomes constricted to form the ureter, a, and this empties into the urinary bladder. The urine then flows down the tubes into the pelvis of the kidney, then down the ureter to the bladder.

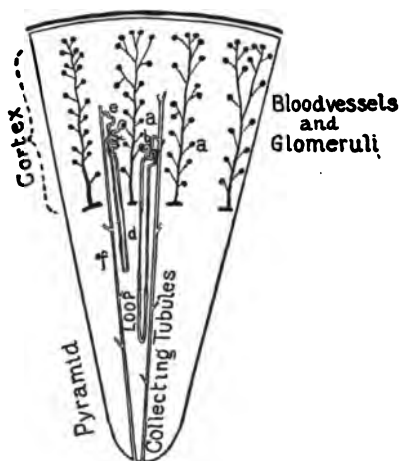


FIG. 77. Scheme of one pyramid. a, the glomeruli arranged like a bunch of grapes on the artery; b, convoluted tubules; d, loop of Henle; f, collecting tubule.

But in man each kidney (Fig. 79) is not a single pyramid but eight or more massed together. In childhood, and in some persons for life, these simple kidneys can be clearly outlined from the lobulation of the cortex. Each pyramid, a, has its own pelvis, here called the calyx, b, and these calyces unite to form the pelvis, c, which by construction becomes the ureter, d. The intermediate spaces, e, are filled with fat.

The two kidneys together weigh about 250 grammes. Sometimes there is but one kidney, or rather, the two are united into one, which, from its shape, is called a "horse-shoe kidney."

The cortex is the most important part of the kidney, because here are all the important structures. It is a zone about 4 to 6 mm. in thickness. In it the glomeruli are arranged on little vertical arteries, like bunches of grapes, and between these rows of glomeruli are the convoluted tubes.

THE AMOUNT OF URINE FORMED.—A normal person voids about 1000 c.c. of urine a day. This amount, of course, varies—even from 800 to 3000 c.c. It depends, first, on the amount of water drunk, and, secondly, on the amount

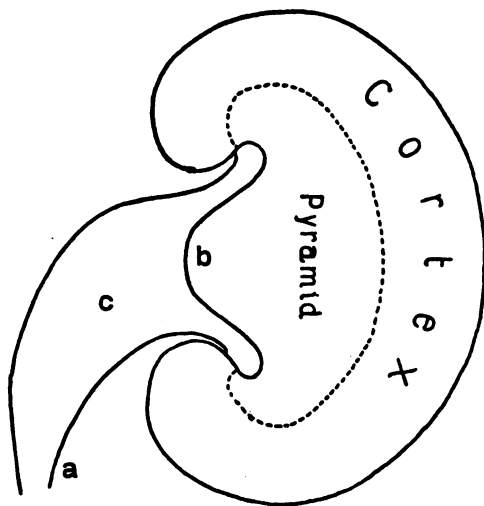


FIG. 78. A "simple" kidney with only one pyramid. The dog, cat, rabbit, etc. have kidneys something like this. a, ureter; b, pyramid; c, renal pelvis.

of fluid lost in other ways, as by profuse perspiration, diarrhoea, vomiting, etc.

Under pathological conditions the amount varies enormously. When the cortex is diseased by the acute or chronic inflammation called nephritis, the secreting cells are more or less affected, and the amount of urine varies almost in inverse proportion to the acuteness of the inflammation. But from the amount of urine alone the condition of the kidney cannot be determined. More depends on what the urine contains (casts, albumin, solids, etc.), than on the amount of water excreted. In the very acute nephritis, there may for a day or so be "anuria," or "suppression of urine;" that is, no

urine at all is formed. When the acute nephritis is not quite so severe, from 50 to 100 c.c. a day are voided, and then amounts increasing according to the improvement in condition. In chronic nephritis the amount is even greater than normal, and in some very chronic cases even twelve litres are voided each day.

The amount of urine varies directly with the amount of blood which flows through the kidneys. This is easy to understand, since the kidney cells can remove the waste from that blood only with which they come into direct contact. In heart-disease with broken compensation, with the venous blood dammed back into all organs, including the kidneys, it is not strange that the urine should be reduced to a small amount per day. A good many drugs, digitalis for illustration, will greatly increase the output of urine in such cases and are therefore called diuretics, but they do so by helping the heart and thus relieving the renal congestion. The amount of urine depends very little on blood pressure, providing this is high enough to keep up the renal circulation.

When there is an unusually large amount of some substance to get rid of, the kidneys will eliminate a large amount of water along with this substance. In diabetes mellitus the patient sometimes has a pound of sugar in the urine, and to keep this dissolved and to get rid of it the kidneys excrete much water, from three to even forty litres in a day. After acute fevers, typhoid especially, the patient may void from three to twelve litres of urine a day. It is supposed that here also the explanation is an increased output of solids, perhaps ashes which have accumulated during the fever. It is thought to be a good sign.

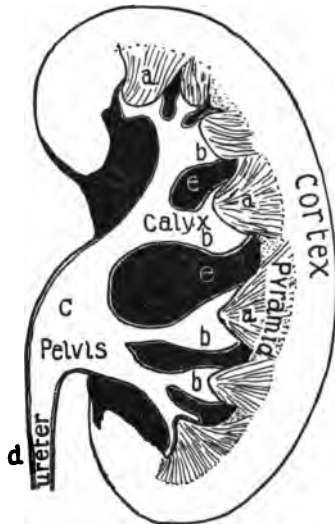


FIG. 79. Diagram of a human kidney. a, pyramid; b, calyx; c, pelvis of the kidney; d, ureter; e, space between calyces, filled with fat.

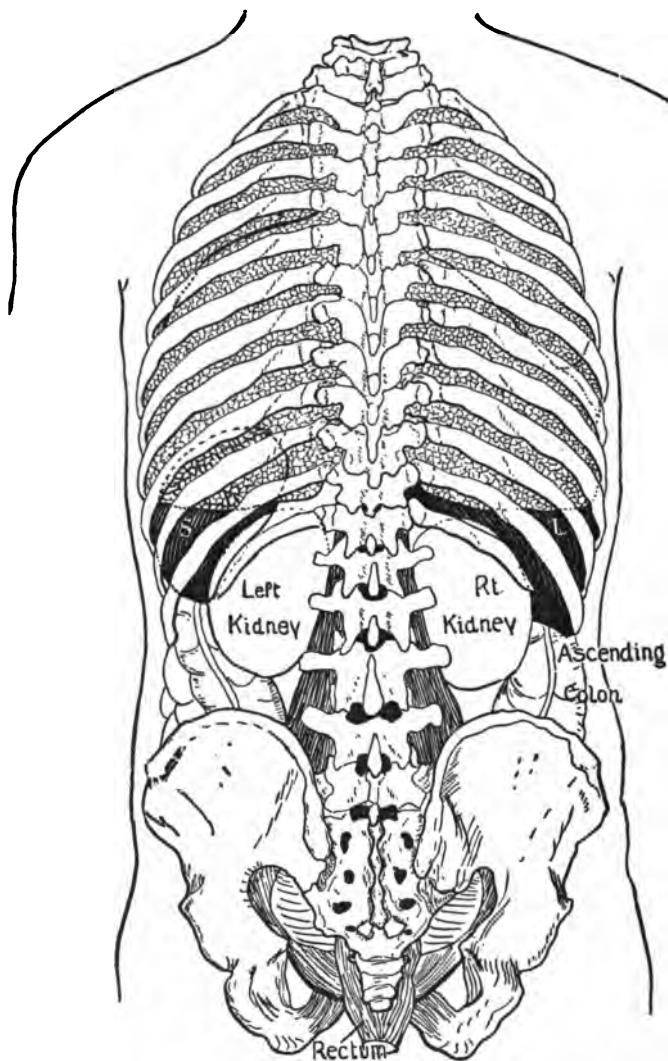


FIG. 80. The abdominal organs, rear view. *S*, spleen. *L*, liver.

Diabetes insipidus is a disease with an enormous output of very dilute urine, even 20 litres a day. It is not due to an increased amount of any solid, and we suppose that, owing to some disease, the kidneys can eliminate only a very dilute urine; and this means an increased amount of water.

In certain nervous conditions, as hysteria, or after epileptic convulsions, the person may void a great deal of water.

Diuretics are drugs which increase the flow of urine. Cardiac diuretics, as digitalis, act on the heart, but some act directly on the renal cells and stimulate them to work harder. Some foods do this—as apples, coffee, etc.—for some persons. Some drugs are diuretics, as caffeine and diuretin (a urea compound). Certain salts act as diuretics because they are foreign to the blood. The kidneys will, therefore, at once excrete them and at the same time considerable water “to wash them out.” This is true of the potassium salts, which we give as potassium citrate, or tartrate in “cream of tartar lemonade.”

The determination of THE SPECIFIC GRAVITY of the urine is sometimes now a part of a nurse's work. By the specific gravity of the urine is meant the weight of a given amount of urine as compared with the weight of the same amount of water. A litre of water weighs at a certain temperature 1000 Gm., and thus 1000 is taken as the standard of specific gravity. Suppose now you dissolve in water a lot of urea, sodium chloride, etc., etc. The weight of the water will be increased, but its volume will increase very little. Measure now just 1000 c.c. of this fluid. Suppose its weight to be 1015 Gm. Then the specific gravity of this fluid is 1015. When we say “The specific gravity of urine is 1025,” we mean that one

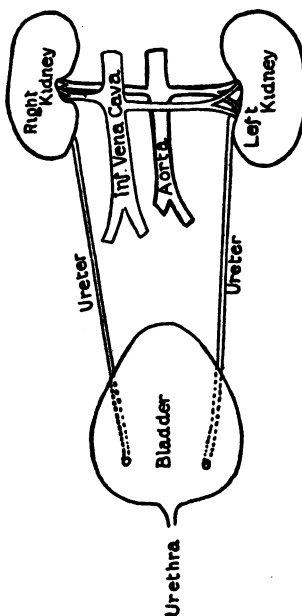


FIG. 81. Diagram of the urinary organs.

litre of it will weigh 1025 Gm. More accurately, the "unit" of specific gravity is 1 instead of 1000, and the specific gravity of the fluid just mentioned is 1.025; but in the case of urine it is more customary to write it "1025." One says, "ten twenty-five," but *never* writes it 10.25; this would mean that

the urine was heavier than iron (sp. gr. 7.7), and almost as heavy as lead (sp. gr. 11.4).

The specific gravity is measured by a "urinometer," (Fig. 82), a glass bulb, *A*, with a weight, *B*, at the bottom, and a long stem, *C*, on which is marked a scale. This scale is so marked that when the bobbin is dropped into water it sinks till the mark 1000 is just at the surface. When put into heavier fluids the bulb will not sink so far, more of the stem will stick out, and on the scale the specific gravity can easily be read.

One must remember that the urine creeps up a little on the sides of the cylinder, *d*, and also on the stem, *e*, forming the "meniscuses." To get the correct reading the eye should be on the level of the line, *f*, and look

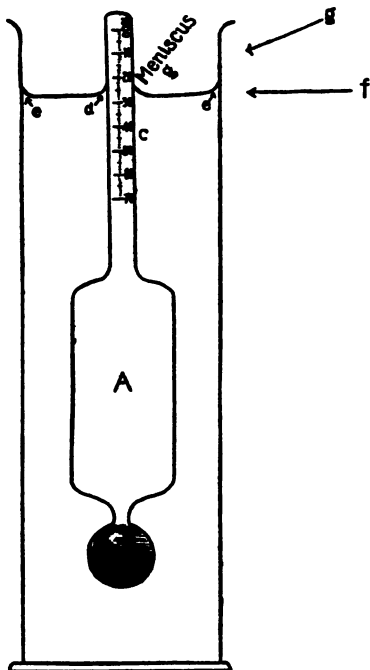


FIG. 82. Urinometer. *A*, the bobbin. *B*, the weight. *C*, the stem. *d*, the meniscus on the stem; *e*, the meniscus on the walls of the glass vessel; *f*, proper line of vision; *g*, improper line of vision.

across under the surface of the fluid, thus avoiding the meniscus. One cannot read correctly looking down from above, as from *g*. The cylinder used should be tall and narrow, and stand on a flat table. The bulb at the time when the reading is made should not touch the side of the glass. If there is foam on the surface of the urine it is easily removed with a piece of filter paper. Each instrument is accurate at but one temperature, usually "room temperature," about 65° F. On

very cold or very warm days the readings will be a little too high or too low. (The specific gravity varies one point for every 3° C. For instance, if the specific gravity of urine whose temperature is 20° C. is 1021, that of the same urine at 11° C. would be 1024; at 26° C., 1019.)

In testing specific gravity only samples of the mixed, total twenty-four-hour amount should be used. That is, all the urine passed in the twenty-four hours must be saved, all mixed together, and a cylinder full of this tested. The character of the urine varies very much from hour to hour, and very unusual figures may be found if single specimens are tested. Normally the specific gravity of the total day's urine is between 1015 and 1020, but that of a voiding, after a heavy meal containing little fluid, may be 1040, and that of a voiding after the ingestion of large amounts of fluid or of some food which acts as a diuretic may be even as low as 1003. If one should find the specific gravity of the total day's urine 1040, it would strongly suggest diabetes mellitus; to find it under 1005 would suggest chronic Bright's disease or diabetes insipidus. A normal person's different voidings of urine could easily give in the course of the day these two readings, but mix up all the urine voided during the whole day and the reading would probably be between 1015 and 1020.

If we wish to know about how much solid matter is voided during the day, we multiply the last two figures of the specific gravity by 2.23; this gives the number of grammes of solids in one litre. This multiplied by the number of litres will give the amount of solids for that day. Supposing that on one day the person voided 2500 c.c. of urine, and that the specific gravity of a sample of the total mixed urine was 1018. Then he eliminated $18 \times 2.23 \times 2.5$, or 100.35 Gm. of solids, during that day.

The specific gravity of urine will depend on the amount of water and also on the amount of solids it contains. One must know both the amount of urine and its specific gravity to judge of a case. Supposing Mr. A. voids 1000 c.c. of urine with a sp. gr. of 1030, and Mr. B. 3000 c.c. with a sp. gr. of 1010. These urines are quite similar. But suppose Mr. A. voided 1000 c.c. at 1030, and Mr. B. 3000 at 1030. Their conditions might be very different.

In nephritis the specific gravity is lower than one would expect, for the kidney cannot easily excrete the solids. In diabetes mellitus even while there is polyuria the specific gravity often runs to from 1025 to 1045 or even to 1060. In diabetes insipidus it may run from 1001 to 1005, and the amounts of urine be very large.

THE REACTION of the urine when first voided is nominally acid. That is, if blue litmus paper is dipped in the urine its color will be changed to red. That is because we eat much food (meat) whose ashes are acids. The ashes of vegetables are alkalies, and hence herbivorous animals void an alkaline urine.

The reaction should be tested just as soon as it is passed, for if allowed to stand even a few minutes on a warm day it will soon be alkaline. The multitude of bacteria which at once get into the urine and rapidly multiply there break up the urea, which itself is neither alkaline nor acid, and set some ammonia free, and this, being a strong alkali, soon more than counteracts the acids which were there. An alkaline urine has a bad odor, in which the odor of ammonia is especially evident. Sometimes even the fresh urine is alkaline. This occurs especially when bacteria are growing in the pelvis of the kidney or in the bladder, and these germs break up the urea before the urine is voided. Sometimes immediately after a meal during which much very acid gastric juice was secreted into the stomach, the urine will, for a few minutes, be alkaline; the reason is that so much acid was taken from the blood by the gastric mucosa that the blood is left very alkaline.

THE COLOR of the urine demands little attention here. The coloring matter of normal urine comes ultimately from the blood. The hæmoglobin of the worn-out red corpuscles is removed from the blood by the liver. Its iron is split off and saved; the rest becomes bilirubin, the coloring matter of bile. In the intestine this is slightly modified, and some of this "hydrobilirubin" is reabsorbed and excreted in the urine as "urobilin." The higher the specific gravity, the deeper the color, as a rule. Diabetes mellitus is an exception, for here the color is pale. In some anæmias the urine is pale because so little hæmoglobin is destroyed, in others it is dark because so much is destroyed. The color depends also on

the food we eat and the medicines we take, and these may produce startling colors, as green or black or deep blue. If there is bile in the urine, which happens in most cases of jaundice, it is best recognized not by the color of the urine, but by that of the foam, for a yellow foam in nearly every case means bile. When a little blood is present the urine is smoky; that is, it is turbid and has a blackish-red tint. After carbolic poisoning and in some cases of constipation the urine is even black.

THE TRANSLUCENCE of the urine is often important. Fresh acid urine is clear. Soon there appears a feathery cloud, the "nubecula," which is the mucus washed by the urine from urinary passages. If the person has not been drinking much water, but has been eating considerable meat, and the day is cold, a remarkable appearance is soon present. The urine becomes very milky, and a heavy layer of white, pink, or very red sediment settles in the bottom of the vessel. It seems great in amount. It is called "urinary gravel," "brick-dust sediment," etc., and usually frightens the person considerably, particularly as several patent-medicine advertisements give this as a sign of serious disease. But this red sediment is the "urate sediment"—that is, a sediment of the salts of uric acid, which are present in every urine, and are deposited whenever the urine is a little concentrated and the day cold. Although especially common in the urine of fever patients, it means nothing at all abnormal in the kidneys. If this urine is warmed, this sediment at once disappears, and the urine becomes perfectly clear.

Only acid urine is clear. When it becomes even slightly alkaline, the phosphates separate out, forming a white sediment, and the fluid above is soon turbid with the enormous number of bacteria growing there. This is the "phosphate sediment," all of which except the part due to the bacteria is at once dissolved if enough acetic acid is added to change the reaction back to acid. This sediment is present in the freshly voided urine if this urine be also alkaline (see page 164), and is present of course in the fresh urine of all herbivorous animals. If the urine is clear when voided, no later appearing sediment has any importance. When urine freshly voided is not clear, but deposits at once a sediment which will not disappear on warming or on the addition of acetic acid, the sediment does indeed mean something. It means usually

either blood or pus or casts, and is called an "organized sediment."

It is very important for the nurse to know how to prevent these changes in the urine, since the examination of a decomposed specimen is difficult and untrustworthy. The urine should be kept in perfectly clean and tightly corked bottles. A few crystals of thymol or gum camphor will often keep it in good condition, or, better still, about a teaspoonful of chloroform, or a few drops of formaline. Much depends on what use the doctor is to make of the urine. If the chemical examination is most important, chloroform is best; if the microscopical, formaline. Whatever preservative is used, the urine should be kept, if possible, on ice.

It is also of value to know how to make clear a specimen turbid with bacteria, for filter paper is of little use. The best method is to add to the urine infusorial earth (called also *Kieselguhr*), about a tablespoonful to 400 c.c. of urine. The urine is then stirred up well, and filtered through ordinary filter paper. The germs are held mechanically by the earth.

Diseases of the Kidneys.—URÆMIA is one of the most dreaded of conditions. It occurs almost always in connection with, or as the result of, kidney disease, and is an "auto-intoxication," the symptoms of which are partial unconsciousness or even coma, with repeated convulsions, and usually, in a few hours, death.

We know that the patient is being poisoned; the symptoms and the autopsy findings prove that. We suspect the poison to be some substance which the kidneys should, but do not, remove, but what that poison is no one yet knows. Formerly it was thought that the accumulating ashes of proteid metabolism, and especially urea (hence the name), were to blame, and this explanation is natural since uræmia is inevitable if for any reason the kidneys cease to perform their function. Uræmia develops if by operation or disease both kidneys are destroyed, or when stones block both ureters. But in very many cases of uræmia careful study of the urine indicates only a slight nephritis, and after death the kidneys themselves sometimes show much less disease than those of other cases of Bright's disease which had no uræmic symptoms. Yet many cases of Bright's disease die in a uræmic condition,

and perhaps all would die thus, did not acute diseases carry off these patients before Bright's disease can do its worst.

Every nurse should know the early signs of uræmia. In the great majority of cases uræmia begins with one or all of three symptoms—headache, drowsiness, and vomiting. Begin active treatment at once, and the uræmia may disappear, for a while at least. But sometimes the onset is sudden. The person may have enjoyed excellent health, and never suspected he had kidney trouble, when suddenly he falls in a convulsion, or is found unconscious, or becomes insane, with dangerous delusions. He may be maniacal and try to kill others, or he may be melancholic and attempt suicide. Such patients are sometimes carried to an insane asylum rather than to a hospital. But the more common onset is seen in patients who have suffered from nephritis. They gradually become drowsy with headache, and vomit. Convulsions follow—sometimes slight, sometimes severe, and often identical with those of epilepsy; the respiration becomes Cheyne-Stokes in character; unless active treatment is successful the coma deepens till death.

Treatment must be prompt and active. Strong purges, even croton oil, are immediately given; the patient is put in a sweat bath once or twice a day. These measures make intestine and skin do some of the kidney's work. Since some, at least, of the poison causing the uræmia is in the blood, we can by bleeding (usually about 500 c.c.) remove a part of it, and by injecting into the same vein a large amount of physiological salt solution we can dilute what remains and thus in some measure diminish the intensity of its action. Often the patient regains consciousness, and the immediate danger is over. The further treatment is that of severe Bright's disease.

NEPHRITIS means inflammation of the kidneys. But inflammation is not the whole story; its results on the kidney, and the effects of poor renal action, are quite as important. In acute nephritis the kidney is inflamed, large, swollen, congested; its cells are injured by the poison causing the trouble, and hence cannot do their work properly; an exudate of blood-plasma, red corpuscles, and leucocytes escapes from the capillaries and infiltrates the kidney substance, with the result that many of these renal cells die and are replaced by

scar tissue. In very chronic cases (and many think some of these are not due to real inflammation) the renal cells slowly die, either because of some poison, or because they do not get enough food. When these cells die (see page 45) their place is taken by a hardier but a useless tissue—scar tissue, and in time the kidney may consist of little else. This process of the degeneration and death of renal cells, and of the increasing of connective tissue, is most conspicuous in chronic nephritis, although perhaps in all cases of nephritis there is present a slight active inflammation which furthers the destructive process. Bright's disease is the term commonly used for almost any disease of the kidney, but it is correctly applied only to the most chronic cases of nephritis.

Acute nephritis, or acute inflammation of the kidneys, is, probably, always due to some poison which directly inflames these organs. This poison may be a drug taken in too large amounts, as turpentine, potassium chlorate, and carbolic acid. These chemical poisons are excreted by the kidneys, which are injured in the discharge of their duty. Or the poison (and this is the most common cause) may be that of germs. Sometimes pathogenic germs are localized in an organ distant from the kidney, as the lung, and the poisons they produce are carried around in the blood and excreted by the kidneys. In some cases the germs settle in the kidney itself and there liberate their poison. This explains the nephritis which develops as a complication of scarlet fever, smallpox, acute tonsillitis, typhoid fever, diphtheria, septicæmia, and abscess. In other cases poisons which injure the kidney are formed by diseased organs. The nephritis of bad burns, and perhaps of some extensive skin disease, may have some such origin, although here also germs may be the cause. Perhaps the commonest cause of acute nephritis, except in that large number of cases which are complications of other diseases, is exposure to cold or wet. Just why and how a nephritis results is not clear, but we know that the poisonous germs are always waiting to attack, and that the exposure lowers the resistant powers of the kidneys. Ordinary acute tonsillitis, "a simple sore throat," is now considered the starting-point of many cases of acute nephritis. The tonsils seem to be the "portal of entry" through which many germs enter our blood.

The symptoms of acute nephritis are very variable. This is a good illustration of a dangerous disease which may progress with serious results without the person's knowing he is sick, until sudden death, or, months or years later, the symptoms of chronic nephritis make the condition only too evident. But the patient usually notices early that both ankles are a little swollen, the face pale, and the eyelids a little puffy. There may or may not be headache, and pain in the back. He may continue in active life until suddenly he is so ill that he must seek treatment. Or, the symptoms (but not necessarily the renal condition) may be very severe and he may suffer from headache, nausea, vomiting, pain in the back, dizziness, chills and fever, and such weakness that he cannot walk. In other cases a uræmic convulsion is the first symptom.

The urine will be scanty; there may be none—"anuria"—for a day or two. But usually the patient passes at first from 50 to 200 c.c. daily of highly colored, cloudy urine with a specific gravity often of from 1020 to 1025, and with a thick sediment of blood, pus, and casts. This urine contains large amounts of albumin. As the patient improves, the amount of urine increases, the sediment and albumin diminish. As the patient gets worse, the anæmia and dropsy will increase, and uræmia may develop. In other cases the patient seems to get well, but the disease continues insidiously, and chronic nephritis is the result. Sometimes, perhaps oftener than we think, the patient gets entirely well.

The treatment must be energetic, even if the patient feels practically well. Its rationale is to rest the kidneys, remove the disturbing cause, and give the kidneys a chance to recuperate. The patient must stay flat in bed for a long time. Any muscular exercise, even moving around in bed, increases the work of the kidneys, and even when the body is in the upright position the circulatory condition of the kidneys is not so conducive to rest as the horizontal.

Since the function of the kidneys is to excrete the ashes of food, the diet given should be one producing few ashes, and those the least irritating in their nature. The ideal diet is milk in small but sufficient quantities. Starvation does not rest the kidneys—quite the reverse. A starving person lives on himself so long as his body contains anything to live

on, and consumes first the stored-up sugar, then the fat, then the protein of the muscles; and the ashes of muscles are more irritating to the kidney than those of milk. One would suppose that a fat and carbohydrate diet would be best, since the ashes of these are eliminated through the lungs. But the body must daily have some fresh protein with which to repair its worn protoplasm. Again, many fat and carbohydrate foods, such as bread, nuts, and vegetables, contain mineral salts which irritate the kidneys. Beef extracts and many soups, must be carefully avoided. Beef extract is made by soaking out of muscle all that can be extracted, and this is really the ashes, which, had the animal lived, would have been removed from the muscle by the blood, and from the blood by the kidneys as constituents of urine. Hence some beef extracts are practically urine. They have little or no nutritive value. For some patients they are very valuable because excellent stimulants, but in nephritis they should be avoided. The kidneys can be much relieved by forcing the skin and bowels to do some of their work, and so purges and sweat baths are given.

Since it is less irritating to the kidneys to excrete much dilute than a little concentrated urine, the patient is given considerable water, and very mild diuretics, such as cream-of-tartar lemonade.

As the patient improves his diet may be varied by a little bread and butter, and even a little meat once a day; and iron may be given for the anæmia. After the albumin has been long absent he may sit up, and later walk. But in the majority of cases the albumin does not disappear, and the patient must plan for the life of a chronic nephritic.

Subacute Nephritis. Chronic Parenchymatous Nephritis.—This form of Bright's disease is one of the most severe if we are to judge it from its symptoms. It attacks young persons especially. It certainly is an acute condition, but it lasts so long and its acute symptoms are of such a grade that sub-acute is the better term. It was called parenchymatous nephritis in that old nomenclature which sharply differentiated the diseases of the parenchyma of an organ (in this case the kidney cells) from those of the interstitial tissue (the supporting structures of connective tissue, the blood-vessels, etc.). Now no such sharp distinction is allowed.

The kidneys of these patients are larger than normal—even three times the normal size—and the cortex, instead of being about 6 mm. in thickness, is in extreme cases even 12 mm. The renal cells are swollen, and full of granular debris and fat. The pyramids are much congested. Here and there throughout the kidney small hemorrhages are seen. There is considerable increase in connective tissue. The urine is at first a little decreased in amount or normal in quantity, but as the case improves it may be much increased, even to 6 or 8 litres a day. It is dirty yellow in color, very turbid even when fresh, and contains very large amounts of albumin. Large numbers of casts of all kinds, of renal epithelial cells, of pus-cells, together with some red blood-corpuscles, settle as a thick sediment. The normal constituents of the urine are diminished, as the diseased kidneys cannot properly perform their functions.

When very ill these patients present a striking picture. The skin has a very pale, pasty color; the whole body sometimes, and always the face, lower extremities, and dependent parts of the body, are swollen with dropsy. Often the finger can be pushed fully an inch into the water-logged skin of the legs. Water collects in the abdominal cavity also—"ascites"—and greatly distends the abdomen. It collects in one or both pleural cavities—"hydrothorax," "water on the chest"—and the patient is short of breath, and must sit upright ("orthopnoea"). It may collect in the pericardial sac—"pericarditis with effusion"—and the patient is in consequence very short of breath, and cyanotic, and has a weak pulse, especially during inspiration.

Many ask why this oedema of the skin (dropsy), and effusions into the body cavities, should develop when the kidneys are excreting even abnormally large quantities of water. One explanation, which may be partly if not wholly true, is that the kidneys are not able to excrete all of the salts, especially the sodium chloride (common table salt), and so this accumulates in the body, especially in the tissues under the skin. But this salt requires considerable water to keep it in a sufficiently dilute solution, and hence holds water there also. Later, when the kidneys get rid of the excess of salts in the blood, the blood can take up this salt retained in the tissues, and with it the water, and the oedema disappears.

Another partial reason is that the heart in nephritis is never at its best, and one result of the poor circulation is the accumulation of fluids in the body cavities. The reason why we mention this "salt theory" of dropsy is that the nurse may understand why these patients are ordered a "salt-free diet," of special bread, well washed butter, and milk treated with various chemicals.

The pulse is hard to compress, which indicates a high blood pressure; the vessel walls already show considerable arteriosclerosis, and hypertrophy of the heart has already begun. The patient often has severe diarrhoea and may vomit frequently. He often is actually on the verge of uræmia.

The causes of this form of nephritis are many. Sometimes it begins as an acute nephritis which refused to respond to treatment, but more often the acute trouble was not evident enough to be treated at all. Or, a patient with scarlet fever, or malaria, or one of many other acute fevers did have a little swelling of the ankles, but no attention was paid to it; he did take a little longer to get well, but was encouraged to get up and be active; his urine after the fever had always a trace of albumin, but no one examined it, so that no one knew of his latent, insidious Bright's disease. Now, however, years later, the debts of this negligence must be paid, and the patient suffers from an illness in which there is little hope. Of course some cases in spite of the best of treatment will develop in just this way, but careful treatment and continuous care will greatly reduce the danger. If one is watchful this condition may be recognized while still quite latent. The person is never quite well, but has gradually failing health, loss of weight and strength, and gets paler and paler. Evenings he may notice his feet and ankles a trifle puffy, and sometimes his face looks swollen. He complains of failing appetite and increasing dyspepsia. He may notice that he must rise at least once every night to pass his water. Then urine examination will show the tell-tale albumin, casts, etc.

The last and perhaps most important cause of chronic parenchymatous nephritis is the habitual use of alcohol, even in small amounts.

The prognosis is bad. A few cases will improve, and the nephritis assume the chronic form next to be described. These patients will, perhaps, enjoy fair health for many years, but

this is the best for which we can hope. The great majority slowly get worse, and die in from one to two years, often in a uræmic condition.

The treatment of these cases is exceedingly important, and the nurse's share especially so. The patient is kept in bed, propped up, and made as comfortable as possible. The diet is limited, chiefly to milk. Some variety of foods is desirable, as the long and limited milk diet will in time add its injury to the condition. The patient is encouraged to drink as much fluid as possible, and in any form. The collections of fluids must be drawn off by tapping whenever the patient is much distended or very short of breath. If he rapidly gets very short of breath, is, perhaps, a little delirious and cyanotic, if the pulse during inspiration is very weak or even loses a beat or so, the pericardium must be examined, for it may need to be tapped at once. The nurse should watch for these symptoms, and for those which suggest uræmia.

Of drugs, diuretin or theocin is given to stimulate the kidneys, and possibly digitalis to aid the heart. The patient must be kept freely purged, and whatever medicines will add to his comfort are justified; for the course is long, the condition serious, and the prospect bad. If uræmia develops, that requires its own special treatment. Should the patient improve, the rule then is to let up on his rigorous treatment very gradually, for the longer the time taken in convalescence, the better the prospect.

Chronic Interstitial Nephritis; Cirrhosis of the Kidney.—By chronic interstitial nephritis is meant a very chronic nephritis in which much of the renal tissue is gone, and its place taken by scar tissue (Fig. 83). There is usually present a slight acute, as well as the chronic, nephritis and flare-ups of this acute process are greatly to be feared. It is a condition of middle or elderly life.

In a few cases chronic interstitial nephritis follows directly as the result of the subacute nephritis, but this is rare. Most of the cases are the result of arteriosclerosis. The renal cells are exceedingly sensitive, and when, as the result of arteriosclerosis, their blood supply is limited, they degenerate and die, and scar tissue, of no use whatever to the body, takes their place. In time there will be surprisingly little real kidney tissue left. In many cases this disease does not follow

either a preceding acute nephritis or arteriosclerosis, but develops as a primary disease. The kidneys of some persons are their "weak organ" from birth, sometimes as an inheritance, and in the strain of life give out first. Overwork, both muscular and mental, and long-continued worry, which keeps blood tension high, are very hard on even normal kidneys, and in time may produce this disease. Some persons injure their kidneys by continued overuse, as does the man

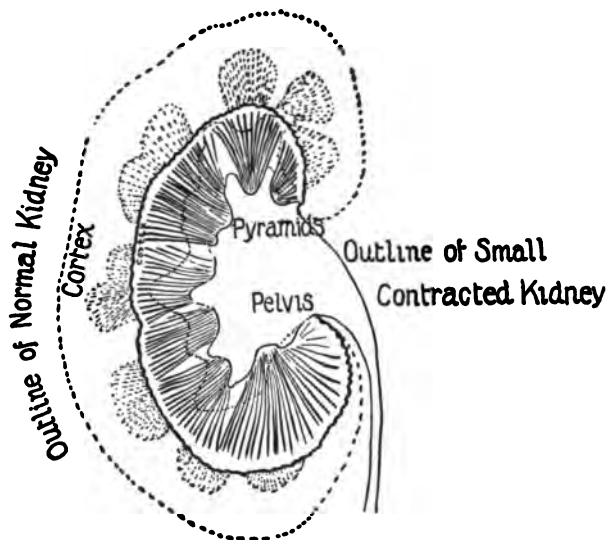


FIG. 83. Chronic interstitial nephritis.

who habitually overeats, especially if he is gouty, or who uses considerable alcohol, or is constantly absorbing poisons, as is the careless artisan who works with lead.

The result of this disease is that the kidneys are reduced even to about one-fifth their normal size. Their cortex shrinks to a layer one or two millimetres in thickness, and in some places is entirely gone. The surface of the kidney is rough, for the renal tissue disappears in irregular patches, and the bands of scar tissue by contracting pull in the surface in places. The glomeruli have, very many of them, disappeared, and the convoluted tubes also. One wonders that the renal tissue which is left can nearly protect the body.

The symptoms are very variable. Some patients with very severe grades of the disease have, for a long time, no symptoms at all. They may discover their condition as the result of an application for life insurance. The first intimation others have is a sudden uræmic convulsion. Or, he has plenty of symptoms, but not of renal trouble; they will more likely suggest heart or blood-vessel disease. If he is carefully examined it is likely that the heart will be found considerably hypertrophied, the arteries very sclerotic, the blood pressure high, and hence the pulse hard. Why in nephritis, especially chronic but also acute, there should be so high blood pressure, even to a point twice the normal, causing arteriosclerosis and a hypertrophied heart, is not known. One theory is that certain ashes of food which the diseased kidneys fail to remove properly from the blood stimulate the small arteries all over the body to contract. This would enormously increase the work of the heart, since it would have to pump against this increased peripheral resistance. Another idea now in vogue is that there are but few glomeruli left, and that the heart is stimulated to pump as much blood as possible through these few, which requires very high pressure. These two ideas may both be true. But the result of high blood pressure is arteriosclerosis of all the arteries, and enlarged heart, and these produce symptoms in many organs.

One of the most common symptoms of chronic nephritis is a slight increase in the amount of urine. The first sign of this increase in amount often is the increased frequency with which a patient must rise at night to urinate. His feet may be slightly swollen at night, but never much, unless an acute exacerbation of the nephritis flares up. He may have any of the symptoms of arteriosclerosis (see page 44); he does not feel well; he loses weight and strength; he suffers from shortness of breath, terrible headaches, and trouble with the eyesight, due to the frequent retinal hemorrhages. At any time, especially after overexertion, the symptoms of uræmia may appear.

The urine will be increased in amount, usually 3000 to 4000 c.c., but sometimes even to 12,000 c.c. a day. It is pale in color, clear, of low specific gravity—from 1005 to 1012. It contains but a trace of albumin, and that is sometimes absent, and so few casts that they are difficult to find. The treatment

is simply to avoid aggravating the condition and to spare the kidneys as much as possible. This condition is apt to last from 10 or 15 years, and the patient will insist on remaining active. The cause if known must be removed. He must eat lightly and drink no alcohol at all. He must refrain from all severe work, physical or mental. Colds must be avoided. The skin and bowels deserve especial attention, because they can relieve the kidneys of so much work. The heart

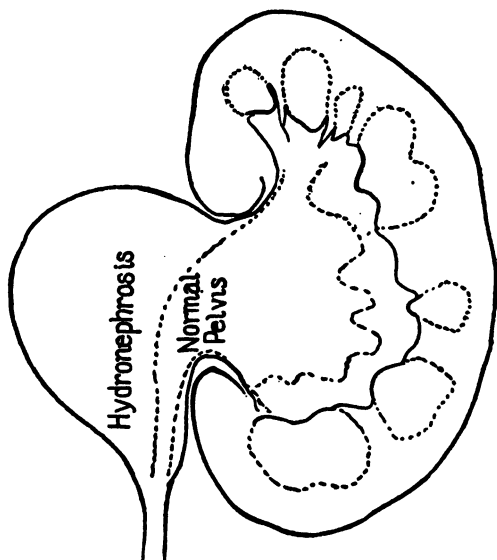


FIG. 84. Hydronephrosis.

condition must be watched and treated. On the whole, the rule of life for this patient is "temperance in all things."

HYDRONEPHROSIS.—The pelvis of the kidney is the dilated sac (including the calyces) into which the urine from the pyramids is poured, and which by contracting to a small tube becomes the ureter. The pelvis is a very thin-walled sac, whose inner surface is lined with the same kind of mucous membrane as lines the ureter and bladder.

When the ureter is obstructed, the dammed-back urine (and the secretion of the mucous membrane) will distend the pelvis of the kidney and the calyces (Fig. 84). Since there is

no inflammation, the fluid is clear. This condition is called "hydronephrosis." If bacteria now lodge there, soon, instead of a clear fluid, pus will fill the sac; the condition is then called "pyonephrosis." The obstruction may be due to a renal stone which was in the renal pelvis, but was swept into the small ureter. If it cannot pass through, it completely plugs this tube. Or it may be due to a tumor which has formed in some other organ and which presses on the ureter, or to bands of scar tissue—the result of an abscess or inflammation near the ureter—which by contracting pinch across the ureter. Or the trouble may be due to an unusual position of the kidney, for often this organ is movable and may twist and kink the ureter. When the ureter is blocked, the fluid constantly accumulating there may distend the pelvis and its calyces, cause by its pressure atrophy of the kidney, which may become spread out into a thin shell; or, if the pelvis is only a little distended, atrophy of the kidney will begin at once. If, however, the obstruction is incomplete, or is intermittent—that is, if the fluid accumulates for a few days, escapes, and then accumulates again—the pelvis may stretch more and more till it almost fills the whole abdomen. The patient may scarcely know of his trouble, or may have rather severe pains as the fluid accumulates. The treatment is to find out the cause, and, if possible, to remove it. If it is a tumor, or bands of adhesions, this is not difficult. If the cause is a "floating kidney," this may be supported by a proper pad and binder, or, if necessary, suspended by an operation.

FLOATING KIDNEY.—During the last few years "floating kidney" has been one of the bugbears of the popular mind, and to it have been attributed all kinds of pain. The kidneys are often easily felt since they are nominally movable, sometimes very much so, but the word "floating" means much more than this. The kidneys are held in place by their fat capsule, and for this reason persons who have suddenly lost considerable weight often have a movable kidney. This condition is much more common in women than in men, perhaps because their clothes fit more tightly around the waist, and in mothers, because of the relaxation of the abdominal walls. The right kidney is often more movable than the left.

There may be no symptoms at all from a movable kidney; also, a kidney may be truly floating without a symptom; but let a nervous woman suspect she has this condition, and, even though the kidney be as fixed as normal, the symptoms will often appear at once, and be distressing enough—pain in the side, dyspepsia, and a whole gamut of nervous disorders.

Dietl's crises (though rare) are especially interesting. These paroxysms of very severe pain, with nausea, vomiting, chills, fever, and collapse, seem to be due to a floating kidney which rotates enough to twist its ureter and blood-vessels.

The treatment of a mild case (and most cases are mild after the patient is somewhat reassured) is to increase the patient's weight, for a few extra pounds of fat in the abdomen are better than silver wire for suspending a floating kidney. The nervous condition of the patient must be treated. If necessary, a well-fitting binder with a pad can be adjusted to hold the kidney in place. If very necessary, an operation may be justifiable, but probably will not be as successful as would a well-conducted neurasthenic-cure.

PYELITIS is a condition deserving especial attention because of its frequent yet unrecognized occurrence. The word means "inflammation of the pelvis of the kidney" (see page 157). The walls of the pelvis are thin, and are lined internally with a mucous membrane which continues down the ureter to the bladder. Inflammation of this mucous membrane of the pelvis is pyelitis. Usually the condition affects but one kidney, but in some cases both.

When inflamed this mucous membrane swells, the surface cells peel off, the blood-vessels become congested, and pus pours out into the pelvis and even fills it. The condition is then called "pyonephrosis." Often tiny blood-vessels rupture into the sac. Soon the ureter becomes plugged, and the sac becomes distended to even large size. Then the kidney begins to atrophy, the pyramids to flatten, and later the whole kidney is but a part of the wall of the sac, and no true kidney substance is left. When the disease is chronic practically all the mucous membrane is destroyed, and then the pelvis is like any abscess. This abscess may rupture. In other cases active pus formation ceases, and the pus dries down to a clay-like mass, in which lime salts are deposited, till the whole is a mass of almost stony hardness.

The urine at first is cloudy because of the large quantities of epithelial cells, mucus, pus, and blood in it, but often also because it is alkaline and the phosphates are precipitated. But sometimes the urine voided on one day is perfectly clear, and we don't suspect any kidney trouble, then, on the next (often on the day when we don't examine it) about one-half of it consists of pure pus. The explanation is that most of the time the ureter from the diseased pelvis is blocked, and only the clear, normal urine from the good kidney, which now does the work of both, reaches the bladder. Then the pus escapes from the diseased side, and the diagnosis is easy enough. Later, when one kidney is destroyed, the other will increase in size and do the work for both.

The causes of pyelitis are many. Perhaps an irritating drug like turpentine can inflame the pelvis, but this is doubtful. In nearly all cases germs are the cause. One must remember that germs (and some of them quite virulent ones) are almost constantly getting into the body, are carried around in the blood stream. Some are eliminated by the kidneys (probably the majority are then dead), the rest are destroyed in the body. When there is in any part of the body a disease due to germs, as abscesses, typhoid fever and tuberculosis, the kidneys are continually removing large numbers of these germs from the blood—so many that sometimes the fresh urine is cloudy from them alone. As a rule, the kidney does not itself suffer, so far as we can see, but when its own resistance is poor then these germs may cause local renal trouble. The kidney's resistance may be lowered by various conditions—general poor health, fevers—as typhoid—simple over-exertion, a cold, some local renal trouble—as Bright's disease—a stone in the pelvis, a local area of tuberculosis on a pyramid, a renal cancer, a twist in the ureter, some tumor pressing from the outside, and possibly an irritating drug. In another very important group of cases the inflammation starts in the bladder and travels up the ureter to the pelvis.

The symptoms of pyelitis vary greatly. There may be pain in the back, but this is rare. There may be no symptoms; this occurs especially in the cases complicating fevers, and the patient merely is a little sicker than before. When the condition is not a complication of a pre-existing fever the

patient will have failing health, become pale, lose weight and strength, not feel well, have some fever. Some cases of pyelitis are treated throughout the whole course for typhoid fever, because the patient looks "typhoidal;" others are treated for malaria, because there are repeated severe chills and fever, with profuse sweats; others are told they have "internal abscess," because they have the hectic fever without sweats; some patient "did have a little fever, probably bilious fever, with typhoidal symptoms, but now he is all right," even while a most active pyelitis is progressing.

The diagnosis is made from repeated, systematic, urine examinations, and, even though the urine is normal, the doctor should be sure normal urine is flowing from both ureters. If on cystoscopic examination none is seen to come from one, a catheter should be passed up into the kidney to make sure there is not a pocket of pus there. If pus is found, it is examined for tubercle bacilli, and an X-ray picture is taken to see if the pelvis contains a stone.

The treatment will depend on the case. Nothing can be done medically, except to treat the patient as for nephritis. In the case of women, the renal pelvis can be repeatedly irrigated. The cases which are complications of acute fevers will get well. On other patients the surgeon should operate. If these are let alone, they may subside, they may even get well—though with the destruction of one kidney; but generally the renal abscess will be the centre of a general infection, and abscesses will develop all over the body.

RENAL CALCULUS. STONES IN THE KIDNEYS. NEPHROLITHIASIS.—As mentioned on page 151, the urine contains many minerals which we find in nature in the rocks. Normally, in the urine these minerals are in solution, but under some conditions one or more of them may separate out and form stones of considerable size and hardness. These stones are made of uric acid, oxalate of lime, phosphates, or of one of the many organic ashes. Why stones form is not well understood, but evidently a nucleus is necessary, and bacteria and dried mucus often furnish this nucleus. The stone sometimes grows slowly till it has filled the pelvis of the kidney, and each calyx, and is a perfect mould of these. It then looks like a piece of coral, and is called a "coral calculus," or a "dendritic stone." Sometimes the stones reach the size of

a pea or become a little larger. Then they may be swept into the ureter and passed in the urine. Some patients pass stone after stone, even several hundred in number, suffering terrible colic each time. Perhaps more often the stones are passed before they are big enough to make any trouble, for we often find small ones about the size of the head of a pin. The stones are most common in the young and old, and are more common in some localities than others. Buda Pest is noted for the number of children who have them. Some counties in England are called "stone counties" because so many persons are thus afflicted.

The effects of the stones are various. In the case of a large dendritic stone, the kidney is very certain to atrophy early, and only a mass of scar tissue may be left. The stones which remain in the kidney usually cause no pain, but sometimes they cause a dull ache in the back, and occasionally severe paroxysms of pain. They may cause pyelitis with all its symptoms and results. A small stone may make no trouble at all, unless it is swept into the ureter by the current of urine or drops in because of some change in the position of the body. If very small it is passed without trouble; if too large to pass, it may block the ureter, causing hydronephrosis; but if it is about the size of a pea, it slowly makes its way down to the bladder, causing the well-known renal colic.

When the journey of the stone from renal pelvis to bladder starts there is a sudden, agonizing pain in the back, usually on the affected side, but sometimes on the other, and this pain runs down in the side, usually to the genitals, sometimes so the inner side of the thigh. It is terrible in intensity and continues until the stone either stops in the ureter or reaches the bladder, lasting seldom less than one hour, sometimes a whole day. There may be a chill and high fever with the pain. Often the patient vomits violently; he perspires profusely; collapse may follow. Suddenly the pain ceases; the stone has probably entered the bladder. But the stone may be so large that it stops in its journey, occluding the ureter. The result will depend on the other kidney, for without operation the kidney of which the blocked ureter is the outlet is doomed. If the other one is diseased, the outlook is bad; if not, it is good.

With the renal colic there is usually some blood in the urine, although a severe hæmaturia seldom occurs. Some pus will be present if infection complicates the case.

The behavior, during the colic, of the other kidney is interesting. Sometimes it ceases to excrete urine; thus "reflex anuria" is supposed to be due to the nervous control of the kidney. Sometimes it excretes great quantities of dilute urine; this polyuria is also attributed to the reflex nervous control.

During the colic hot applications are often very agreeable—a hot-water bottle at the side, hot drinks, and especially a hot bath. Usually large doses of morphia or chloroform are necessary.

Between attacks the patient should, above all else, eat very temperately, remembering that the constituents of stones are the ashes of meat, and should drink large quantities of water. There is a great amount of humbug about the best waters to advise, and about drugs guaranteed to dissolve stones, but both waters and drugs, so far as we know, are all practically useless. Unless the stones are passed readily, by far the best treatment is their surgical removal.

TUMORS OF THE KIDNEY.—The kidney may be the seat of various tumors, including cancer, and some of these reach huge size. The patient often feels the lump, and suffers dragging pains on the side of the kidney. When the tumor is malignant he very often has hæmaturia; in fact, blood in the urine not easily explained by some evident disease should always suggest this condition.

"CONGENITAL CYSTIC KIDNEY." **CYSTIC DISEASE OF THE KIDNEY.**—This condition probably dates back to infancy, although it may not be evident till adult life. Both kidneys are affected, but usually one more than the other. The kidneys may be huge and fill the entire abdomen. All the natural appearance of the kidney is lost, and the tumor consists of a mass of cysts, from the most minute to those of bean size. The kidney tissue itself is the seat of an advanced chronic interstitial nephritis. There is often blood in the urine.

CHAPTER IX

DISEASES OF THE NERVOUS SYSTEM

FOR EVEN a superficial understanding of nervous diseases so accurate a knowledge of the anatomy of the nervous system is necessary that any discussion of them in these lectures is almost impossible. We shall, therefore, speak of only a few of the familiar diseases and of these mention only the most prominent symptoms.

THE CENTRAL NERVOUS SYSTEM consists of the brain and spinal cord. These are safely enclosed in bony cases, the brain in the cranium, the cord in the backbone. They are wrapped up in the membranes, or "meninges." (See page 282.) The brain consists of cerebrum, cerebellum, pons, and medulla. It weighs about 1500 Gm., or a little over three pounds.

The cerebrum is the largest portion of the brain. It consists of two halves of almost equal size, united at their lower borders by the "pons," or bridge. Each half, or "hemisphere," of the cerebrum is hollow, and the cavity, the lateral "ventricle," is filled with fluid. When because of disease this fluid increases in amount, it must, of course, distend the cerebrum. If the patient is a baby, and the bones of the skull are not united, each of these ventricles, instead of containing about 50 c.c. of fluid may hold 500 c.c. or more. Then the hemispheres are so distended that they resemble thin bladders, and the head is swollen to huge size. This terrible condition is named "hydrocephalus" or "water on the brain." A somewhat similar condition occurs in adults, but since their skulls cannot distend a very little extra fluid will cause severe symptoms, headache, vomiting, blindness, etc., and more will cause death.

The cerebrum consists of gray matter which is spread on the surface in a thin layer about one-eighth of an inch thick, the "cortex" (Fig. 85, *a*), and white matter, *b*, which lies between cortex and ventricle, *c*. The gray matter is the

important part; this contains all the brain cells, the "batteries" of the nervous system. The white matter consists almost entirely of long fibres, the "wires" which connect the cells with the organ, or muscle fibre, etc., which that cell controls. These fibres are so fine that a strong microscope is needed to see them, yet some of them are fully two feet long. If the cerebrum had a smooth surface, there would not be cortex enough, and so its surface is folded in many "convolutions" with deep crevices, or fissures, between. In this way the amount of gray matter is considerably increased without increasing the size of the brain. The larger convolutions are fairly constant and serve as excellent landmarks in finding the various areas of the brain.

Fig. 85 a.

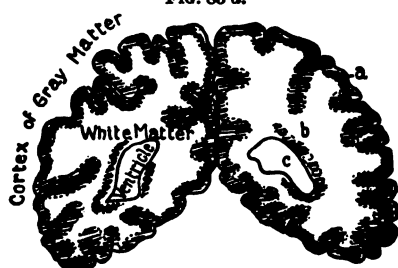


FIG. 85 a. A cross section through a tip of the brain showing the arrangement of the gray and the white matter. The gray matter is external and near the ventricles, the white is internal.

Fig. 85 b.



FIG. 85 b. Cross section of the spinal cord showing the arrangement of the gray and white matter. The gray matter is internal and the white is external.

The brain cells of the cortex look alike, but have very different duties to perform. There is, for instance, on each hemisphere a vertical band of cortex, the "motor area," which governs the voluntary movements of the muscles of the opposite half of the body. This can be accurately mapped out. We know the exact spot where lie the cells in which originate the voluntary movements of the muscles of the face, the thumb, the hand, the arm, the body, the leg, etc. If we wish to move a muscle it is these particular cells which send the stimulus down along their fibres. Or, stimulate these cells with an electric current, and the same muscles contract. If, therefore, a man has paralysis of certain muscles, and this paralysis is due to disease of his cortex, we know before we open the skull almost the exact spot on the cortex where we shall find that trouble; or if, as sometimes occurs after fractures of the skull, the person has convulsions of certain muscles, or convulsions of the whole body which

always begin in certain muscles ("Jacksonian epilepsy") we can tell just where to look for the sliver of bone, etc., which is pressing on the cells which govern these muscles. The motor area of each hemisphere is rather large, but the fibres from it converge and pass out of that hemisphere into the pons collected into a small bundle, the "internal

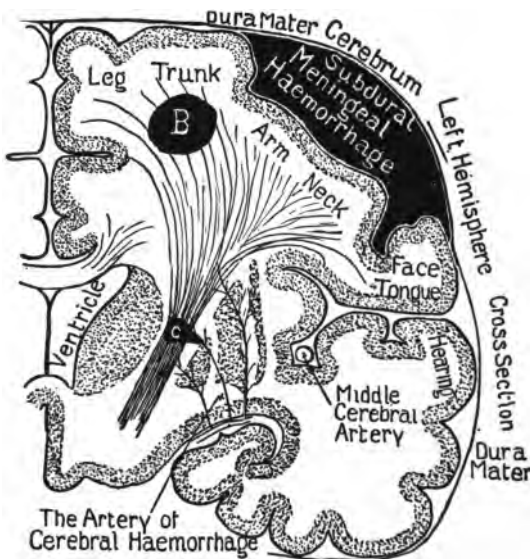


FIG. 86.—A cross-section of the left hemisphere of the brain through the motor area. The subdural meningeal hemorrhage compresses the brain over the arm-neck area. There is no real destruction of brain substance, but paralysis would result from pressure. *B*—Subcortical hemorrhage. This hemorrhage does destroy brain substance. Although a smaller hemorrhage than the subdural, it produces possibly more paralysis, since it cuts fibres which have converged somewhat. *C*—Hemorrhage into the internal capsule. This explains the common "stroke" of paralysis. Although the hemorrhage is small, it causes total paralysis of the right side of the body, since it cuts all the fibres in the internal capsule.

capsule." This bundle then passes through the pons into the cord, crossing as it does so with that from the other side. It runs down the cord, as the "pyramidal tract" (Fig. 86). A very small injury to this bundle will paralyze many more muscles than will a much larger injury to the cortex itself. It is just as in a central telephone station, one blow of an axe can sever all the wires at the point where they leave the building, while a similar blow on the switch-board would sever but few. The ordinary case of a "stroke"

of "apoplexy" followed by paralysis of one-half the body ("hemiplegia") is usually due to a tiny hemorrhage from a blood-vessel in this "capsule." A very much larger hemorrhage nearer, or at the cortex, might paralyze one or a few muscles, but hardly a whole half of the body. Hemiplegia may be due to the rupture of a miliary aneurism on the tiny artery running in the internal capsule, "the artery of cerebral

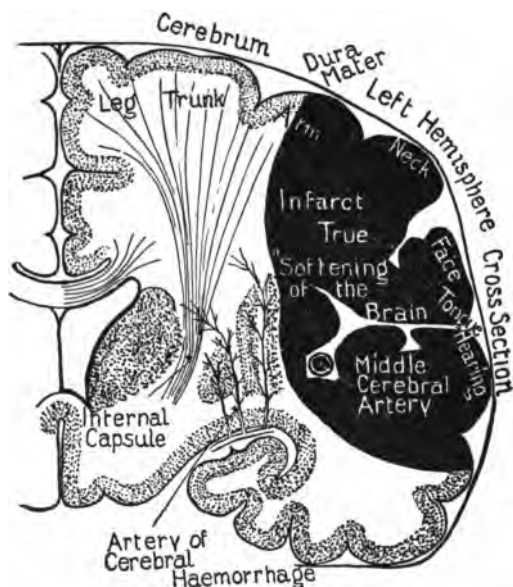


FIG. 87.—A cross-section of the left hemisphere of the cerebrum through the motor area. The middle cerebral artery is represented as plugged by a thrombus. That part of the brain (black area) supplied with food by this artery has died (infarction, or "softening"). This patient would have paralysis of the right side of the face, tongue and neck, and part of the right arm; he would be unable to talk and understand what was said to him.

hemorrhage" (see page 48), or to the plugging of this artery by a thrombus or embolus and the subsequent death of the fibres which it supplies with food. Why this artery, of the greatest importance in the brain, should suffer so much oftener than the others we do not know. Immediately after a shock one whole half of the body as a rule is paralyzed. Then gradually the person recovers the use of certain muscles, usually of those of the leg, often of those of the upper arm, least often of the hand. The reason for this is that the hemorrhage may

actually destroy the fibres to a few muscles only, but temporarily injure all those in the neighborhood, perhaps by the pressure of the blood. As the swelling from the hemorrhage diminishes, these latter will resume their function; but those destroyed, never.

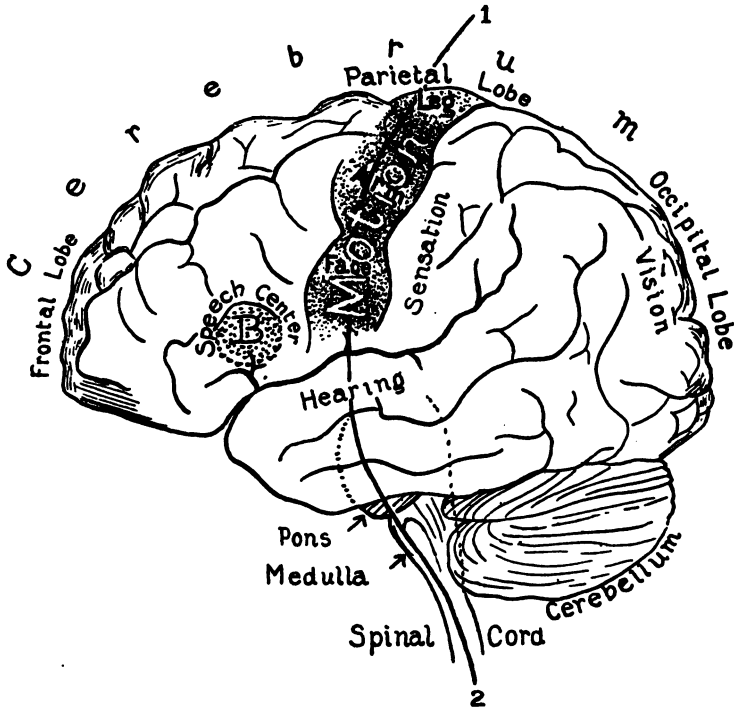


FIG. 88. Side view of the brain. The line 1-2 is the direction of the schematic cross-section represented in Figs. 86, 87 and 93.

In "Broca's convolution" (Fig. 88 *B*) the cells look like those of the motor area, but really have nothing to do with muscles. Broca's convolution is an area on the left hemisphere, of about a square inch in size, and which adjoins the motor area. Here are the cells where are stored up the "combinations" of muscular movements necessary to speak each word. They are not the cells which govern the muscles of speech; these cells are in the motor area itself. Each word, even the simplest, requires for its utterance quite a combination and

sequence of combinations of muscular contractions; not only must the muscles of the vocal cords contract, but those of the throat, of the tongue, of the soft palate and the lips; and of the chest wall as well. To pronounce even a short word is a very complex action. In the cells of Broca's convolution these "combinations" are stored up. They direct the cells of the motor area, and these make their muscles contract at the proper time and with the proper force. This area is so near the left motor area that disease of the latter often affects the former. This is why so many persons who are paralyzed on the right side (due to a lesion of the left hemisphere) are unable to speak; while those paralyzed on their left side almost never have any speech disturbances. In some cases they do, but these are left handed persons whose speech area is on the right hemisphere. (This is one proof that to be right handed or left handed is not a matter of training, nor of habit, but depends on the very structure of the brain itself. Lose the right hand and the left may become fairly proficient, but never very.) When Broca's convolution is destroyed, the person is said to have **MOTOR APHASIA**; he knows the word he wants to say, he understands all that is said to him, he can read, has absolutely no paralysis of the vocal cords, but to produce the sequence of movements of the throat necessary to produce that word he cannot, and if he tries he makes an unintelligible noise. He is in much the same position as the banker who has forgotten the combination of his safe. He may turn the dial as he will, if he does not turn it back and forth to exactly the right figures the safe will not open. And yet the lock is all right and the man is strong enough to turn the dial any number of times he will—the trouble is that he has forgotten the combination.

In other areas are the cells in which are stored up the memory of the way the words which we want to speak, or which we hear, sound, the way they look when written, etc. If these areas are injured, the person has **SENSORY APHASIA**. This patient may be able to talk volubly, but point to a chair and ask him what it is, and he cannot remember the word "chair," although he may use that word frequently in his rambling conversation. Ask him if it is a "chair" and he at once assents. There is no paralysis, and the word is correctly spoken, there is no motor aphasia.

We are all "functionally" aphasic at times. I meet an old acquaintance, but cannot recall his name try as hard as I will. I "know his name well, but cannot remember." I have for an instant sensory aphasia. Again, in talking to Mr. Jones, I unconsciously call him Mr. Smith, and yet am sure I said "Jones." For that instant I have "motor aphasia."

In aphasia due to true brain disease the man may be totally aphasic at first, and yet may in time partially or entirely recover his speech. If so the real destruction of cells and fibres was not as great as we first imagined, but many were for a while rendered functionless, since they lay in the "halo of temporary disturbance" which surrounds a genuine destructive lesion.

There are definite areas on the rear of the brain where end the fibres of the optic nerves. It is by means of these "receiving cells" that we see. Be the eye ever so good, the optic nerve ever so perfect, if these cells are diseased, we are blind. For illustration, my friend is telephoning to me. He may talk distinctly, his telephone may be in perfect order, the wire intact, but if the receiver I hold in my hand is out of order I may not hear a word. So, although my eye, my optic nerve, etc., are perfect, if this cortical area is diseased, I will not see; I have "cortical blindness." Blindness in one eye may be due to disease of any part of that eye itself, or to disease of its optic nerve. Just behind the eyes the two optic nerves unite, then separate, and continue to the brain as the "optic tracts." In each of these tracts is just half of each optic nerve, and so, if one tract be injured, there will be blindness of one-half of each retina. For instance, if the right tract be injured, the patient will be blind on the right half of each retina, and so will see nothing at his left hand with either eye. In a case of blindness we can thus locate the trouble.

There are, also, cortical areas where we smell, others where we hear, etc., and larger areas, especially under the forehead, the office of which we don't know, but diseases of which change a man's disposition or moral character.

The functions of the cerebellum are too little understood by the expert to be studied by the nurse. It is enough to say that disease here causes, among other symptoms, "motor incoördination;" that is, the patient cannot use his muscles accurately; he stumbles, and fumbles, is dizzy and staggers.

The pons is a connecting link between the two hemispheres; from it also starts the "medulla," which is the bulbous upper end of the spinal cord.

Let a TUMOR grow in the brain anywhere and certain general symptoms will follow. These are severe headache, vomiting, usually without any nausea, and changes in the retinae of the eyes due to pressure on the optic nerves and resulting at last in blindness. There are other more local symptoms; paralysis, irritation of the cortex, changes in the special senses, which often show just about where in the brain the tumor is.

The central nervous system communicates with the rest of the body through the peripheral nerves, which are bundles

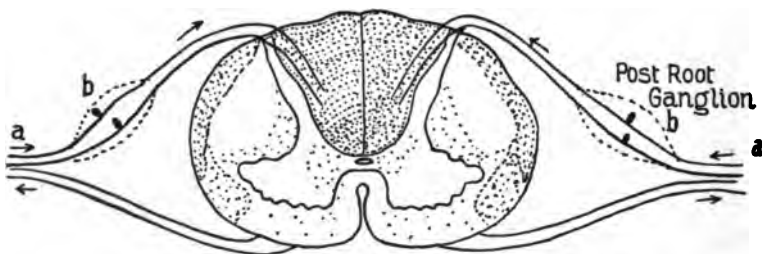


FIG. 89. Cross-section of the normal spinal cord. The dotted areas show the location of the sensory fibers *a*, which run through the posterior root ganglia *b*, to the cord and up to the brain.

of the white fibres, Fig. 89, *a*. Nerves are always arranged in pairs one for each side of the body. Some nerves, called "motor nerves," run from the brain to muscles. Cut these and the muscles they supply are paralyzed. The cells from which these fibres start are in the central nervous system. Other nerves, called "sensory nerves," run from the skin and sense organs to the brain; cut these, and the area of skin, etc., from which they come is made insensible. The cells of these fibres are outside the central nervous system, collected in small masses called "ganglia," Fig. 89, *b*. Most nerves are "mixed" since they contain both motor and sensory fibres.

From the brain there are twelve pairs of nerves, called CRANIAL NERVES. The *first* pair are the "olfactory nerves" or nerves of smell, which connect nose and brain; the *second*, the "optic" nerves, or nerves of sight, connecting the eyes and brain. The *third* is a pair of motor nerves which supply all

but two of the muscles which move the eyeballs; the *fourth* pair supplies one of these two and the *sixth* the other of these two muscles not supplied by the third pair.

The *fifth* is the largest pair of the cranial nerves. It is a mixed nerve, whose sensory portion comes from the skin of the head and face, the membranes of the eyes, mouth, and the pulp of the teeth. Its motor portion innervates the muscles of mastication. Each fifth nerve divides into three large nerves. The first supplies the tear glands and the membranes of the eye and the skin of the forehead and the inside of the

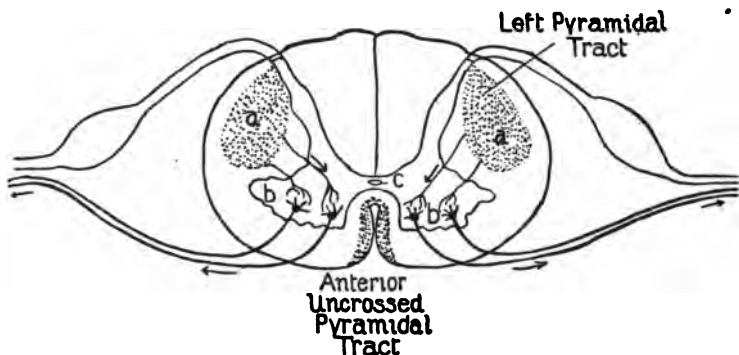


FIG. 90. A cross-section of a normal spinal cord. The dotted areas represent the location of the motor fibres which run from the cortex of the brain to the motor nerve cells which control the muscles. *a*, pyramidal tracts of motor fibres; *b*, anterior horn cells, the nerve cells which directly govern the contractions of the muscles. The function of the anterior uncrossed pyramidal tract is not known.

nose. The second branch supplies the skin of the nose, cheeks, upper lip, lower eyelid, and the upper teeth. The lower branch contains the motor portion and also receives sensory fibres from the skin of the temple, lower lip, lower jaw, part of the tongue, and the teeth of the lower jaw. The fifth nerve is the nerve of FACIAL NEURALGIA, or "tic douloureux." The whole nerve may be affected, or one branch only in which case the patient knows only too well just what that branch supplies. Mild cases of neuralgia are due to trouble in the nerve starting in a decayed tooth, or to some other local disease. Remove this, and the patient at once gets well. But true tic douloureux is due to disease of the nerve itself. It is a disease of middle life especially. In a severe case, the pain comes on in paroxysms of terrible intensity, separated by

periods of remission, which may last from minutes to months, but which grow shorter as the disease progresses. The paroxysms are brought on by cold, by movements of the face or tongue, by a touch on the skin, by almost any stimulus to the head or face. It often starts at one spot and radiates along the branches of this nerve, and even into those of other nerves. The pain is so frightful that many patients commit suicide. Some few cases have recovered spontaneously, but the best treatment is surgical. If only one branch is affected, this branch is exposed at the point where it is nearest the skin, is seized by a pair of forceps and pulled out, the object being to tear out as much of the nerve as possible. The nerve will grow again in time, but the patient has about eighteen months or more of comfort. Then an inch or so more may again be torn out at the same place. In case the whole nerve is affected, or the pain is very severe, the skull should be opened and the ganglion of the sensory portion of the nerve removed. Then the nerve will not grow again, and the cure is permanent. This operation may be thoroughly recommended. After the operation the nurse has considerable responsibility, for part of the patient's face is now insensible, and hence must be carefully guarded from all injuries until the tissues readjust themselves to their new state. The eye especially needs watching. Normally, if a piece of dust gets under the eyelid the pain is sharp and we get rid of the particle as soon as we can; but a patient after this operation will not know the dust is there and an inflammation may set up which may even destroy the eye.

There may be a NEURALGIA of almost any nerve, but those most affected are the nerves of the back of the neck, of the shoulder and arm, of the chest wall ("intercostal neuralgia"), of the small of the back, "lumbago," of the lower tip of the spine ("coccydynia"), the great nerve of the leg, "sciatica," and of the feet. These conditions differ chiefly in the location of the pain. The treatment is the same for all; rest of the affected part, a thorough rest cure of the whole system, and tonics of various sorts, but morphia never, for the habit is easily formed by these patients. The best local treatment is the Paquelin cautery, electricity (the continuous current), the ether spray, belladonna ointment, etc. If severe the only recourse is an operation on the nerve.

The *seventh* cranial nerve, the "facial nerve," runs to nearly all the muscles of the face. When one of this pair is much affected **FACIAL PARALYSIS** ("Bell's palsy") is the result. This paralysis may be due to trouble anywhere along the nerve from cortex to muscles, but most often the trouble is in that part of the nerve which lies in a long canal in the temporal bone. This canal is very close to the middle ear, and if this is inflamed the disease easily affects this nerve also. The commonest cause of facial paralysis is to "catch cold" in the face by exposure to a strong wind, etc. The nerve becomes inflamed (neuritis) and swells. For the nerve to swell where it lies under the skin does little harm, but for the swelling to extend to the portion lying in a canal of hard bone, which is only just big enough to hold a normal nerve, is more serious, for the pressure paralyzes it. In a case of facial paralysis the affected side of the face is smooth, and does not move when the patient frowns, closes his eyes, or moves his mouth, etc. The upper eyelid droops, the mouth is drawn to the good side, since the paralyzed muscles are now limp and do not normally oppose their antagonists on the other side of the face. In eating and drinking the food drops from the paralyzed corner of the mouth. Ask the patient to whistle and he cannot. The cases due to cold practically always get well in time. If seen while getting well one may mistake the side which was paralyzed, for now for a while the paralyzed muscles contract more strongly than do the others and wrinkle the side which formerly was smooth. The treatment is to remove the cause if possible; *e.g.*, to treat the ear trouble. If due to cold, little can be done save to keep the paralyzed muscles in as good condition as possible by electricity and massage.

The *eighth* nerve is the auditory nerve, which connects the inner ear with the brain. The *ninth* is the glosso-pharyngeal, the nerve of the sense of taste and the motor nerve of some of the muscles of the throat.

The *tenth* nerve is the "vagus," or "pneumogastric nerve," the motor nerve of the voluntary muscles of the throat and larynx, the nerve through which the brain governs the rate of the heart-beat. It sends branches to the lungs, but their function we do not know; and branches to the stomach and œsophagus which are important in vomiting at least.

The *eleventh* cranial nerve is the spinal accessory nerve, so named because one of its branches joins the tenth nerve. The other branch is the motor nerve to the sterno-mastoid and trapezius muscles. The sterno-mastoid is the muscle of the neck which is conspicuously placed as it runs from just behind the ear obliquely downward and forward and attaches to the collar bone. The trapezius is the large heavy muscle which runs from the skull behind down the back. Its edge determines the curve of the neck and shoulder. "Wry-neck" or "torticollis" is due to contraction or shortening of these muscles and is often cured by cutting them. Often other deep neck muscles are also involved.

The *twelfth* or hypoglossal nerve is the motor nerve for many muscles of the tongue and throat.

The importance of the brain cortex is strikingly shown in the GENERAL PARALYSIS OF THE INSANE, or "dementia paralytica." We may describe this disease in popular terms as an atrophy of the cortex, probably due to a very chronic meningitis. This disease is supposed to be the same disease as locomotor ataxia, only the latter affects the membranes of the cord, and so injures the white matter on the surface of the cord, while dementia paralytica affects the membranes of the brain and so injures the cortex. At any rate, the membranes of the brain "grow onto the cortex." The cortex becomes thin, and the convolutions small. It is a disease of adult life and especially of men. The earliest symptom is often a change in the character of the patient. He becomes very egotistical, is sure he is the best, or the richest, or the strongest man in the world. He does unaccountable things; may waste his property in wild, impossible schemes. He may commit some foolish, filthy, or desperate act, sin, or crime, and often boasts openly of it. He may have convulsions resembling epilepsy. Then one notices a tremor of the face, slowness of speech, loss of memory, then delusional insanity, then strokes of paralysis. Later he is insane and paralyzed, then later still demented and paralyzed, and then comes death. There is no cure. When the first symptoms are seen a specialist should be consulted, and if the patient has general paresis he should be put at once into an asylum.

The SPINAL CORD is that part of the nervous system within the backbone. It is a cord about eighteen inches long

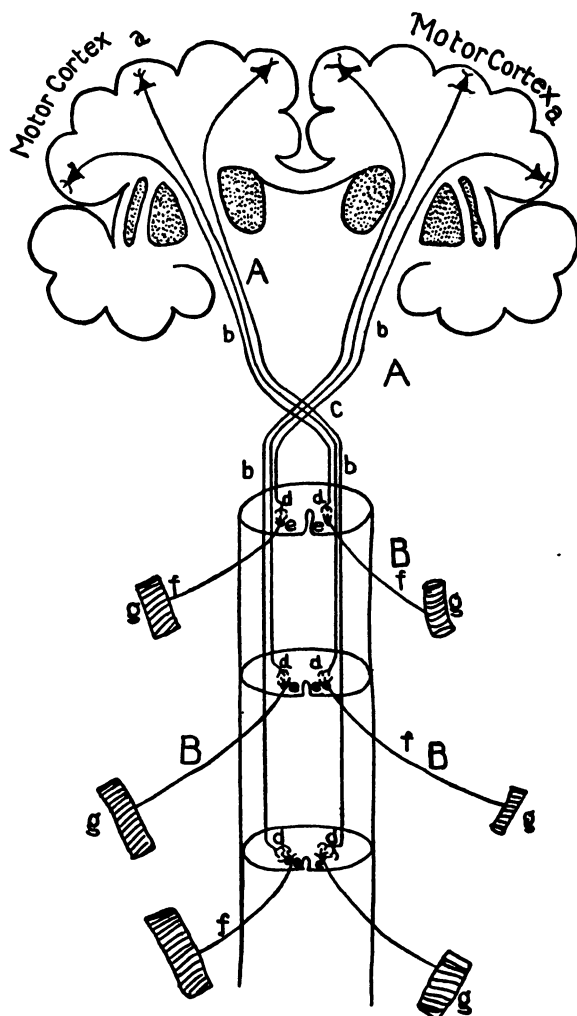


FIG. 91. Scheme of the motor path. *A*, upper motor neuron; *B*, lower motor neuron. *a*, motor cortex, containing the cells of the upper motor neuron; *b*: fibres of the upper motor neuron, which run down the cord and end in the anterior horns, *d*, in contact with the anterior horn motor cells, *e*; *f*, fibres of the anterior horn cells, *e*, which run to muscle fibres, *g*. *c*, decussation of pyramidal tracts.

and approximately the size of a man's finger. It is a direct continuation of the medulla oblongata. Like the brain it consists of gray and white matter, but while in the brain the gray matter is external and the white internal, in the cord the gray matter is in the centre and is surrounded on all sides by the bundles of white fibres, the sensory fibres running up to the brain, and the motor fibres down from the brain. The cord gives off thirty-one pairs of "spinal nerves." These nerves are all "mixed." Those leaving the right side of the cord supply the muscles, skin, and organs on the right side of the body; those of the left side the corresponding muscles, etc., of that side of the body.

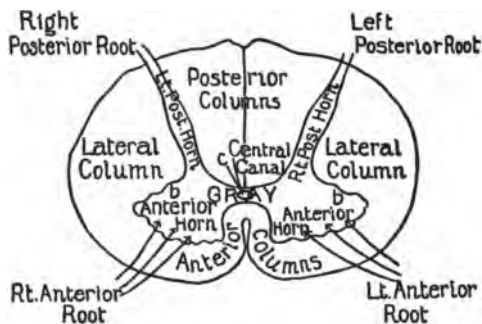


FIG. 92. Cross-section of the normal spinal cord.

We have described the motor area (Fig. 87) of the brain cortex. Here are the brain cells which "generate" the nervous impulses by means of which the will makes a muscle contract. These impulses travel down the long fibres of these cells. These fibres pass through the medulla to the cord in a bundle of motor fibres, called from its shape the "pyramidal tract," (Fig. 91, b). In the medulla this bundle or "tract" crosses that from the other side, c, and then travels down the cord on the opposite side from that on which the cells lie, but on the same side as the muscles for which their impulses are intended. At a certain point in the cord, d, a fibre leaves the white matter of the cord, enters the gray matter, and comes to an end. It never passes out of the cord into a spinal nerve. The motor fibres of the spinal nerves are all fibres of cells, e, in the gray matter. These fibres, f, run from these cells into the spinal nerve and its branches, each to its own muscle fibre, g.

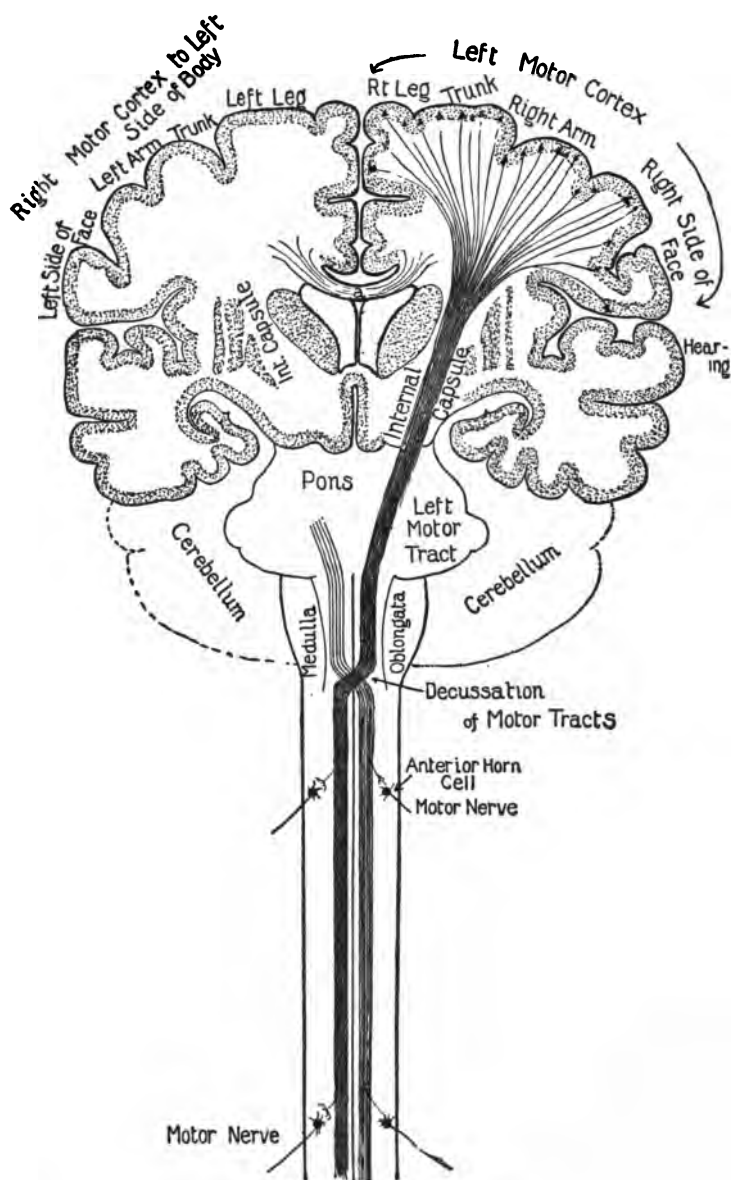


FIG. 93. Diagram of the motor tract. The line 1-2 of Fig. 88 shows the direction of this schematic section of the central nervous system.

From what we have stated it is seen that the brain controls each muscle through a combination of two cells with their fibres. The cell in the motor area, and its fibre running down the cord is called the "upper cell" or more correctly "upper neuron" (Fig. 91, A), and the cell in the cord whose fibre runs to the muscle, called the "lower cell," or "lower neuron," B. The motor nerve of a muscle is a bundle of thousands of these fibres, each coming from one motor cell in the cord, each going to one or a few of the thousands of muscle fibres of which the muscle is a bundle.

Paralysis of a muscle may be due to trouble in the nerves anywhere from brain cortex to the muscle. Suppose the nerve

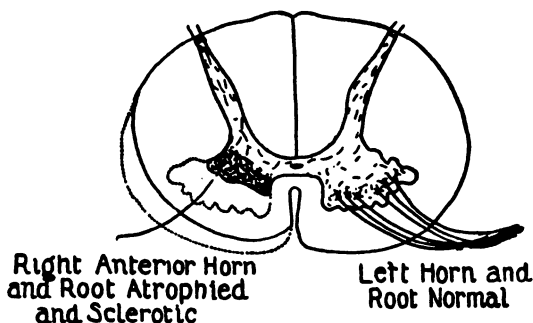


FIG. 94. Cross-section of the cord of a case of anterior poliomyelitis. Note that the right horn has atrophied, its cells have disappeared, and that the anterior root of the spinal nerve is shrunken.

is cut somewhere between the spinal cord and the muscle (Fig. 91, *g*). The muscle will be paralyzed; that is, the man cannot through any effort of will move it. Also it will take no part in "reflex movements." But that is not all, for the muscle becomes limp and wastes away, that is, it atrophies, since this lower motor cell governs not only the movements, but the health of the muscle. Soon a new nerve will probably grow. But now suppose that not the nerve fibres but the cells from which they come, are destroyed. Then the nerve will not grow and the muscle will never be useful again. In anterior poliomyelitis of infants we have a good illustration of this last form of paralysis.

ANTERIOR POLIOMYELITIS is an inflammation of the spinal cord and results in the death of certain of the motor nerve cells there. These cells are in the anterior, or front part, of

the gray matter of the cord, hence the word "anterior;" "polio" means gray, and "myelitis" means inflammation of the spinal cord (or "marrow"). These motor nerve cells die, and consequently their fibres also die. If the fibre is cut or injured a new one will grow out from the cell, but a dead cell is never restored, and the muscle fibre it supplies will always be paralyzed. This disease occurs especially in infants under three years of age, seldom in adults. The cause we do not know. Some think it is a blood-vessel disease; some say it is caused by a germ, and it certainly does occur in epidemics. But the mothers are seldom in doubt as to the cause, and usually unjustly blame the nurse for "letting the child fall," or "letting it catch cold." The child has an acute fever for a day or so, and then the mother notices that a part of the body is paralyzed. Sometimes a whole limb is paralyzed, sometimes only a few muscles of a limb; sometimes parts or the whole of two limbs, etc., but there is no "system" or "order" to the distribution of the paralysis, as there is in diseases of the upper motor neuron. At the end of the first day the paralysis is at its height. Then there is a gradual improvement, since many of the cells were not destroyed but only temporarily injured. These latter will regain their function, but those destroyed, never. Then the child grows. If the whole of a muscle is paralyzed, it atrophies. If only a part of the muscle, then it will long be weak. The result is wasting away of some, the partial wasting away of others and weakness of more. That limb or part of limb will throughout life be shorter and smaller than its mate. All of our muscles have antagonist muscles, and these normally balance each other. If now one becomes weak or paralyzed its antagonist will pull unopposed, and so bend the joints and cause serious deformities. Hence it is that many club-feet, etc., arise. There will be no disturbance to the sensations of the limb, since only motor cells were affected. The diagnosis is easy. If a child or adult has an arm, leg or foot shorter, smaller, and weaker than the other, and the joints of this limb are loose or "flail-like" so that they dangle and are of little use, the chances are that the condition is due to this disease. If it dates from infancy and is "due to a fall," one may be sure. The treatment is very important. Some of the muscles are not entirely paralyzed, and will get much stronger if carefully

massaged and exercised by electricity. This must be begun early and done systematically, for after twelve months there is little use in treatment. The distressing deformities can now be cured by the surgeon, who will transplant the whole or part of the tendon of a healthy muscle to take the place of the useless muscles or muscle.

But suppose it is the upper motor neuron which is "cut," (or in any way destroyed). Then a very different state of affairs exists in the muscle. It will be paralyzed as far as the "will" goes, but not necessarily for "reflex" (unconscious) movements, for these originate in nerve cells below the motor cortex. It will not atrophy, for the nerve cells governing the growth and health of the muscle are uninjured; it will not be limp, but even more strongly contracted than normal. This paralysis seldom affects a few muscles only, since a tiny injury affects so many fibres, but usually a whole limb or both limbs, or a half of the body.

A good illustration of this form of paralysis is the SPASTIC (that is "stiff") PARALYSIS of infants. This disease affects especially children whose birth was assisted by forceps, or who receive other mechanical injury during birth. There is rupture of a blood-vessel in the meninges of the brain, and the long continued pressure of the blood causes partial destruction of large areas of cortex. When the child should begin to walk, it is noted that the legs especially and the arms also are stiff. Since the muscles which bring the feet and knees together are naturally stronger than those spreading those limbs apart, the legs in this disease are often crossed and walking is by "cross-legged progression;" that is, in each step the leg is moved not only forward but across the other. When both legs and both arms, or both arms, are paralyzed, the disease is called "diplegia;" when both legs alone "paraplegia" (when the arm and leg on the same side, "hemiplegia"). The hemorrhages usually cover much wider areas than the motor area alone, and the children are usually deficient mentally. They commonly have convulsions. They can use their limbs somewhat, but awkwardly, stiffly, and weakly.

But a better illustration of paralysis due to disease of the upper motor neuron is seen in the HEMIPLEGIA OF ADULTS. A hemorrhage, embolus, or thrombus destroys the fibres from

the motor area in the internal capsule. The arm and leg of the opposite side become at once stiff and paralyzed, and the reflexes exaggerated. A better illustration still is the paralysis following a fracture of the spine due to injury, or to a knuckle in the backbone due to tuberculosis. The edges of the bone crush the spinal cord completely at that point. There will be stiff paralysis, "spastic paralysis," of all muscles on both sides whose nerves leave the cord below the crushed spot, and limp paralysis of the muscles whose nerves leave the cord exactly at the crushed place, since the cells of the lower motor neuron are destroyed. There will also be insensibility of the skin below the crush, since the sensory fibres no longer run to the brain.

Another good illustration of upper neuron disease is seen in adults with **SPASTIC PARAPLEGIA**; that is, a chronic stiffness of the legs, due to a gradual degeneration of the fibres in the pyramidal tract. The person notes that his legs are stiff. He walks as if wading through water, taking each step with difficulty and hardly raising the

foot from the ground. During each step the knees practically always touch each other. Later on the muscles are not only stiff, but weak. Try the knee kick and the leg is thrown into what is almost a convulsion. The sensations are unchanged.

In the white matter of the cord one can pick out various bundles of fibres. The pyramidal tract runs down in a definite portion of the cord (see Fig. 90, *a*). There are definite bundles of fibres which convey sensations from the muscles; others which convey the sensations of touch, pain, heat, cold, etc., for each of these sensations has its own "service" of cells and fibres. Some of these tracts do not run in the white matter, but through the gray matter. We can therefore diagnose the position of some lesions by their effects on the various sensations, granting that they do not affect the whole cross-section of the cord. For instance, here is an adult

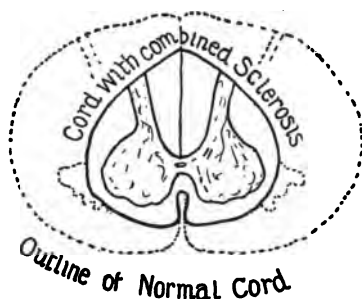


FIG. 95. A cross-section of the spinal cord of a case of combined sclerosis. The white matter especially has atrophied. The symptoms of this patient might resemble those of a case of locomotor ataxia.

who has lost the sensation of heat and cold and of pain, but not of touch in the skin of his hands and arms; he has often scalded himself without realizing it; babies have been killed by being bathed in too hot water by these patients. Hearing such a story, we suspect at once a disease which destroyed the cord only in its centre, and is probably SYRINGOMYELIA (*syrix*, pipe; *myelia*, cord; therefore, freely translated, a cord like a hollow pipe). There is normally a tiny canal down the middle of the cord, Fig. 92, *c*, but in syringomyelia, Fig. 96, this canal, *c*, is much larger than normal, not because the canal is distended ("hydro-myelia," in which case no tissue is destroyed), but because there is destruction of the gray matter

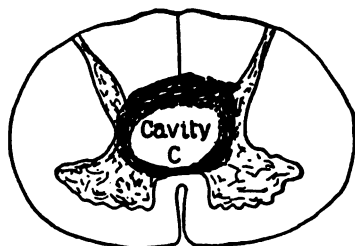


FIG. 96. A cross-section of the cord of a case of syringomyelia. There has been a growth of the "glia" tissue around the central canal, and a cavity, *C*, forming in this tissue has greatly increased the size of the canal.

around this canal. Since the fibres carrying the thermic and pain sensations travel up the cord in the gray matter around this tiny central canal, in syringomyelia these are the ones which are destroyed, and yet the skin feels touch with normal acuity because the fibres carrying this sensation travel up the cord at some distance from its centre. There will also be other symptoms; spastic

paralyses with muscular atrophies, pains, etc.

Again, the fibres which are responsible for "muscle sense" travel up the posterior part of the cord. TABES DORSALIS or "locomotor ataxia" is due to a very chronic posterior meningitis, which destroys these fibres (Fig. 97) of muscle sense, and the result is that the patient cannot control his muscles. The muscles are not weak, and there is no paralysis, the man can contract them with normal force at will, but since the muscle sensations are gone he cannot use them skilfully. This is ataxia, or "loss of order." Notice such a patient as he walks. He watches every footstep to be sure he makes it correctly. As a rule we walk almost unconsciously, the muscle sense guiding the muscle contractions, but this man cannot trust these sensations and must use his eyes. He cannot walk in the dark, since the sight sensations also are

then lost. His skin sensations are keen enough, but lying in bed he often "loses his legs," and, if he cannot see them, he must feel for them to know whether they are straight, bent, crossed or even hanging out of the bed. He stands erect well enough, but, if while doing so he closes his eyes, he falls (Romberg's sign), since then he cannot control the muscles whose constant play holds us in equilibrium. Of course there are other symptoms. He has sudden atrocious pains, especially in his legs, which are well described by their names, "lightning" or "shooting" pains. He has sudden attacks of vomiting, various bowel and bladder symptoms, queer sensations in his feet, certain eye signs, loss of knee kicks, etc. But the symptom we wish to emphasize here is the loss of the muscle sense by means of which we use our muscles skilfully, and by means of which we know without looking exactly the position of each limb and joint. The result of this loss is pathetic. The patient is a strong man, with power enough to move his muscles forcibly, but he has a gradually decreasing

power to control his movements. He first walks along rapidly with long, strong, swinging steps, and a stamping gait, but watching each step; later he uses a cane to keep him from falling, then two crutches, and finally must be supported on both sides, and even then his legs get "all tangled up." The three cardinal symptoms of tabes are: inability to stand upright with the eyes closed; the loss of knee kicks; and the failure of the pupils to contract when light is thrown into the eye.

The nerves themselves may be the seat of disease. NEURITIS means inflammation of a nerve. This may be acute or chronic, of a single nerve, "local neuritis," or affect many nerves, "multiple neuritis." Local neuritis may follow exposure to cold (*e.g.*, Bell's palsy, see page 193) or injury, or the extension of an inflammation from neighboring organs. There is pain along the nerve, it is sore when pressed on, and,

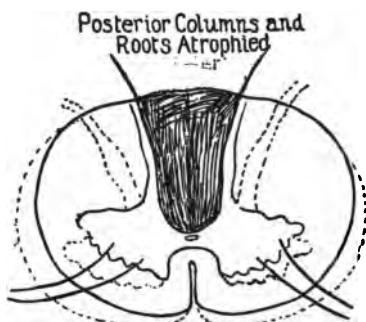


FIG. 97. Cross-section of the cord of a case of locomotor ataxia. The dotted lines show the outline of the normal cord.

since its functions are interfered with, the muscles it supplies will be more or less paralyzed, and the sensations from the skin from which its sensory fibres come will be less sharply perceived.

SCIATICA will serve as a good illustration of a local neuritis. The sciatic nerve is the largest nerve in the body. It supplies nearly all the muscles of the legs and feet with motor branches, and receives the sensory nerves from nearly all the skin of the lower extremities. It starts at the spine in the pelvis and runs down the back of the leg. Neuritis of this nerve may be due to exposure to cold, to a severe wetting, to severe muscular exertion, to the pressure of tumors in the pelvis, and most often to chronic "rheumatism" of the spine with the formation of bony nodules which pinch this nerve. There is intense pain running from the thigh to the foot, a pain made much worse by any motion of the leg which puts the nerve "on the stretch." It is for this reason that the patient keeps his knee bent and walks on his toes. Pressure anywhere over the nerve is very painful. There may be wasting of the muscles. Sciatica is a very difficult disease to treat, since it is so hard to remove the cause. It is very chronic and apt to recur. When severe, the patient may become bedridden. The doctor must first be sure there is no disease of the spine or of the pelvis which presses on, or pinches, this nerve, for if these are removed the patient can get entirely well. Medicines are of little value. Long rest in bed, or warm baths, may cure a case. If necessary drugs or even pure water can be injected directly into the nerve with very good effect. Or, the nerve may be exposed by operation and then pulled to stretch it somewhat—a procedure which often succeeds in relieving the pain.

HERPES ZOSTER, or "shingles," may be mentioned here, although this trouble is not so much an inflammation of a nerve as of its ganglion, which is on the sensory portion of each spinal nerve just where it leaves the cord, Fig. 89, b. The nerve cells of this ganglion become inflamed, hence the sensory fibres of the nerve also become affected. In this disease the patient feels ill for three or four days, then there appears a large crop of blisters, each of which is exactly the same as a cold sore. This crop of cold sores appears in the area of skin from which the sensory fibres of that nerve come.

In fact this is the best way to map out the areas of skin from which the sensory fibres of a spinal nerve do come. Whenever we see a case of shingles we map out accurately just where the vesicles appeared, and in that way have by this time accurately charted the skin. When the spinal nerves running to the chest and abdomen are affected, there is a perfect band of vesicles half way round the body, hence the name "zoster" or "zone." The vesicles themselves do not stay long, but after they heal there continue the distressing burning pains of neuralgia in that nerve, and these make shingles a very painful disease.

MULTIPLE NEURITIS.—Sometimes the inflammation affects many nerves, as a rule the same nerves on both sides of the body, and usually those of the arms and legs. With it there is some pain in the nerves, tingling in the fingers and toes, partial loss of sensation in the skin and of power in the muscles which the affected nerves supply. The muscles most paralyzed are usually the extensors of the wrists and the flexors of the ankles. Ask the patient to hold his hands out horizontally and they hang limply down ("wrist drop"). He cannot lift his toes from the ground ("foot drop"), hence he lifts at each step the whole leg so high that the loose dangling foot cannot drag on the ground (the "steppage gait"). All the muscles of the body may be thus partially or wholly paralyzed, and the patient may die from inability to move the muscles of respiration. As a rule the muscle weakness lasts from one to several months, then is followed by a slow convalescence.

Multiple neuritis may occur as an acute febrile disease following exposure to cold, wet, etc. Then there is fever, headache, malaise, all the symptoms of an acute infection. But the most common cause of multiple neuritis is the steady use of alcohol. For months the patients have neuralgic pains, and tingling in the hands and feet. Then the wrist and foot drop become evident. Very rarely are other muscles affected. These cases practically always get well, but it takes months. Alcoholic neuritis is often accompanied by mental symptoms, a psychosis with delusions in which "time and place" seem utterly lost. That is, the patient is sure he was a few minutes ago miles away, and will tell of events of years ago as if they were yesterday.

Multiple neuritis also follows the infectious diseases, as typhoid, but more often diphtheria. The toxin of diphtheria picks out almost always certain nerves, especially those to the eye and throat. Too much arsenic, either as a medicine, or even that in some beers, can cause it. "Beriberi" (page 323) is a multiple neuritis.

The treatment of multiple neuritis depends on the nurse more than on the doctor. In all cases the patient should rest in bed and take a general tonic treatment. The nerves will take months to recover, since many nerve fibres must be replaced by new ones. In the meantime the muscle fibres may become so weak and atrophied that they are almost useless when finally their nerve fibres have recovered. The secret of successful treatment therefore is to keep the muscles in good health by daily massage, oil rubs and electricity, and then when the nerve is well the muscle also will be well.

The effect of the degeneration of motor nerve fibres on the muscles is well seen in "PROGRESSIVE MUSCULAR ATROPHY." In this disease there is slow degeneration of the lower motor neuron, and often later of the upper also. The motor cells of the cord disappear, hence their fibres in the motor nerves also disappear. The result is a slow atrophy of muscles with at first a fine twitching of their fibres. The hands are first affected. The muscles waste away, lose their strength and dexterity, and finally contract, forming a "claw hand." Later the muscles of the forearm, then of the upper arm waste, and stiffen; then the rest of the body, till, finally, we have the "living skeletons" of the dime museums. It is a disease of adult life. Its cause is not known, nor is any treatment of avail.

CEREBRAL HEMORRHAGE.—This condition, popularly called "apoplexy," a "stroke of paralysis," or a "shock," is usually due to the rupture of a blood-vessel in the brain. The patients subject to these shocks are men with arteriosclerosis, especially those who work very hard and who drink heavily. On the cerebral blood-vessels minute aneurisms the size of a pin's head may develop, or the brain tissue degenerates around a vessel, forming there a little hole called a "lacuna." Both of these are spots along the arteries. The blood pressure in these cases is usually very high and the logical sequence is, sooner or later, rupture. The blood pours through the rup-

tured vessel, infiltrates the tissue, and thus kills much of it. So many cases of stroke are due to rupture of one certain artery that this vessel is named "the artery of cerebral hemorrhage." Unfortunately, this supplies one of the most important spots in the brain, the internal capsule.

Sometimes an artery on the surface of the brain ruptures and then the blood clot gathers under the meninges and presses on the brain. These "meningeal hemorrhages" are due especially to fractures of the skull, or, as in the case of the new born, to injury. Here again one particular artery is most liable to rupture, and that runs over one of the most important parts of the cortex.

EMBOLISM AND THROMBOSIS.—"Softening of the brain" (Fig. 87) is a disease often mentioned, but by that term the lay-mind usually means the gradually developing dementia, the loss of memory, of reasoning power, etc., which accompany old age. Real softening of the brain does occur, but its symptoms are quite different from the above. It is the result of the closure of a cerebral artery by an embolus or thrombus. The brain tissue which depends on this vessel for food dies, and becomes really soft: Emboli in nine-tenths of the cases come from a heart with endocarditis (see page 50). Unfortunately vegetations loosened from a heart valve often travel (because it is the straightest course) via the internal carotid artery, directly to the brain, and pick out the artery to the motor area. The onset of the paralysis is sudden and quite resembles a stroke of paralysis due to hemorrhage.

A thrombus may form in any diseased artery. Its result is quite similar to that of a thrombus due to an embolus except that its symptoms develop more gradually.

If the vessel plugged by an embolus or a thrombus supplies a "silent area," there will be no localizing symptoms, and sometimes no symptoms at all.

The "stroke" due to hemorrhage (Fig. 86) or to embolus, comes on, as a rule, without warning. There is usually sudden and total loss of consciousness with complete relaxation of the arms and legs. In other cases the loss of consciousness is more gradual, while in still other cases the paralysis develops without loss of consciousness. While in coma the respirations are slow, deep and snoring; the cheeks, especially that on the paralyzed side, are blown out at each expiration. One

side usually is perfectly paralyzed (hemiplegia); which side it is, can be recognized even during the coma by raising the arms and letting them fall. The paralyzed arm will fall more like a "dead" limp limb. There may be a convulsion; the eyes are often turned to one side, the side of the brain in which the trouble is. In a few hours (from two to forty-two) the fever begins. The patient gradually regains consciousness, and then the hemiplegia becomes more apparent. The paralysis is, of course, the side opposite to the trouble. It at first usually involves face, arm, and leg; that is, it is a complete hemiplegia, but it may be partial and affect only one of these. The muscles of the thoracic and abdominal walls usually escape (these seem to get nerves from both sides of the brain). When the paralysis is of the right side, there is usually aphasia also (see page 188). The paralysis is usually much more complete at first than it is later, since many fibres are only temporarily injured, and later recover their function. For this reason the paralysis may entirely clear up. The permanent paralysis is usually worst in the arm, and especially the forearm and hand. The leg recovers somewhat and the patient is usually later able to walk, while the face often almost completely regains its functions. The sensations are very little affected, but in rare cases the paralyzed side is very hypersensitive. Later, the paralyzed limbs become very rigid, especially the arms, and contractures result; that is, the elbow and wrist become stiff and flexed in various positions. After any severe shock the patient always has some mental weakness, and is more emotional and irritable than before.

We have spoken chiefly of the results of destruction of the motor area, but of course there are other cortical areas the lesions of which give localizing symptoms; such as blindness of one-half the fields of vision, deafness, abnormalities in smell, loss of skin sensations over half the body, etc. But all these areas added together, including the motor, comprise only a small part of the cortex. The rest of the cortex is called "silent," although changes in character have been known to follow lesion of the frontal lobes.

The early diagnosis of apoplexy is very difficult. A man is found unconscious, breathing heavily and with all his limbs limp. The doctor, and sometimes the nurse, must decide between apoplexy, uræmic coma, diabetic coma, fractured

skull, morphia poisoning, epilepsy, or a simple "drunk," and must do so in a very few minutes. This diagnosis is not easy and mistakes are very often made. The uræmic patients usually have some cedema of the limbs and always albumin in the urine; the drunken man's breath may help, but in a case of one of the other possibilities the chances are some friend has tried to pour whiskey or brandy down the man's throat and over his clothes; the breath of the diabetic coma case has a sweet, "fruity," odor, and urine is rich in sugar; the epileptic had first a convulsion which someone may have seen, and often has a bleeding tongue, but so may the uræmia case; the opium poisoning case developed his coma gradually, etc. But many of these points demand some previous knowledge of the case. The urine should be examined at once for albumin and sugar; the skull for a fracture, the limbs should be raised and allowed to fall to detect paralysis; the eyes examined to see if they both look to one side (brain injury, hemorrhage), etc.

Death in apoplexy usually occurs before the third day. A fall of temperature and return of consciousness on the third or fourth day are good signs. Often the paralysis clears up entirely, but if not within the first month, there is sure to be some permanent loss of power.

The treatment of cerebral hemorrhage, thrombus and embolism depends more on the nurse than doctor. It consists in keeping the patient perfectly quiet in bed, turned on one side if the respiration is hindered when lying on the back. If there is good reason to suspect hemorrhage and the blood pressure is high, the patient is usually bled at once. There is, however, some doubt as to the value of this procedure, and by far the best treatment is to open the skull and to remove the clot. An ice-bag on the head, hot-water bottles at the feet, and a calomel or croton oil purge at once are indicated in all cases. The bladder should be catheterized if necessary. In the treatment of embolus and thrombus there is little to do, save keep the patient quiet. After about ten days after the attack the paralyzed limb should be massaged gently each day, and electrical treatment begun about a month after, the object of both being to prevent the limbs getting stiff in an unfortunate position (contractures).

PARALYSIS AGITANS.—Paralysis agitans, "shaking palsy," or "Parkinson's disease" is a nervous trouble the chief symp-

toms of which are tremor, muscular weakness and muscular rigidity. The cause of the disease and its location in the nervous system no one knows. Perhaps the nervous system of these patients has aged sooner than normal and more rapidly than their other organs, for the tremor resembles that of old age, only much exaggerated. It affects men especially, in particular the hard workers. They often date the trouble back to a severe wetting or exposure to cold, or to excessive muscular exertion. The mental faculties are little impaired. The tremor begins in one hand and arm, then the other, and often finally the head. There is especially a slow turning motion of the forearm and hand and a motion of the thumb against the fingers as of a man "rolling a pill." If the patient gets excited the tremor is worse; when he makes a voluntary motion, it ceases. He can skilfully do the most delicate acts, such as pick up a pin, etc. At the same time the patient's limbs are weaker and more rigid than normal; he stands with head bent a little forward and walks as if in danger of falling on his face. Since its muscles move little the face has very little expression, and is spoken of as "mask like," a feature which can be recognized at a glance.

The only treatment is to improve the general health.

EPILEPSY.—"Epilepsy is a disease characterized by attacks of loss of consciousness, with or without convulsions." That "epilepsy" and "fits" are not synonymous the lay mind can scarcely believe, and yet some of the most dangerous epileptics have few if any true convulsions. Attacks of momentary loss of consciousness without convulsions, "petit mal," have really the same significance as have the most violent convulsions. These patients may feel dizzy and fall, or for a moment stand as if dazed, or make some automatic motion, sometimes a very violent one. These alone may occur at first, but sooner or later the definite convulsions, "grand mal," begin, and then the patient will have both.

The epileptic convulsion is often preceded by an "aura" or warning. This may be a physical or mental sensation, a flash of light, a sound, etc. Then with a loud scream the patient "falls as if shot," making no effort to protect himself, hence often is injured. The body is perfectly rigid, with jaws fixed, hands clinched and legs extended. The muscles of respiration also are contracted and the patient gets bluer and

bluer. In a few seconds begin the convulsive movements, slight at first, then more and more violent. Practically every muscle in the body is violently affected. The tongue is often chewed; the stools and urine are passed involuntarily. After one or two minutes the convulsive movements become less violent, the body relaxes, and the patient lies in deep coma, breathing noisily. The respirations then are chiefly abdominal. He cannot be aroused for a few minutes or even hours; then he gradually awakes. For a time afterwards he is confused and complains of headache and malaise. During this period he may be truly insane and commit crimes that he afterwards does not remember. Some patients seem to have "fits of mania" without any convulsions. The earliest convulsions in an attack occur only at night. The patient wakes up feeling badly, or finds that he has bitten his tongue, or voided his urine in bed, but does not for a long time suspect the true nature of his trouble.

True epilepsy begins usually in childhood and continues throughout adult life. Some patients get well, but their attacks usually began after puberty. The cases which begin before ten years of age seldom get well. For a patient to have frequent attacks means that it is a severe case. In all cases there is sooner or later some mental deterioration, especially in those with frequent fits. Those with maniacal symptoms after a convulsion are dangerous and should be confined in an asylum. While some persons with epilepsy have been intellectually even famous, it is well to remember that in some states epileptics are grouped among the insane. Epilepsy seems to be seldom if ever inherited, but it does occur especially among the children of the neurotic, the hysterical, the insane, and especially of the intemperate.

The treatment of epilepsy is one of the great problems of this age. Bromides are the only drugs which seem to lessen the number of fits at all, and do cure some cases. Sodium bromide well diluted in milk is the least irritating form. In those cases in which the fits cease entirely the drug should be continued for at least two years after the last fit. It is necessary to give large doses and to keep the patient almost saturated with the drug. One stops temporarily when the patient complains of drowsiness, or gastric or cardiac distress.

The diet of epileptics should be carefully arranged since gastric disturbances are common. Complications and errors in diet seem frequently the exciting cause of a fit. They should eat very little meat, not oftener than once a day, while perhaps a purely vegetable diet is the best. The evening meal should be especially light.

Epileptic children should receive kind but firm training, and should be taught some trade which will keep them busy. The present idea favors their segregation in colonies, and this is certainly from the point of view of society the best idea.

During a fit a cork should be placed between the teeth to protect the tongue. The patient should be stretched out on the floor, or put in a reclining posture, the clothes about the neck and chest loosened and he be let alone.

Of "idiopathic" epilepsy we can find no cause on examining the brain; all we can say is there is an irritation of the motor cortex. But in "symptomatic" epilepsy, while there is little if any difference in the fits, there is a discoverable cause, and one which sometimes can be removed by operation. The fits in "reflex" epilepsy stop entirely after the cause is removed. Among children the causes of the reflex fits are intestinal worms, trouble with the genital organs, a foreign body in the ear or nose, etc.

Epileptiform fits occur in various brain diseases, such as a tumor, chronic meningitis, etc., while the fits of uræmia may exactly resemble those of epilepsy. The most common condition which must be excluded is hysteria, but the experienced nurse seldom mistakes. In hysteria the patient never falls as if shot, but is careful to select a spot, and to fall in a manner, that she will not hurt herself. The movements are not truly convulsive, but rather struggling; or the whole body is held rigidly. The persons around must beware or they will get struck or bitten. The hysterical person may scream during the attack, the epileptic never after the onset. The epileptic fit is over in a few minutes, the hysterical often not for hours, but it may be terminated at once, sometimes by pouring ether over the skin, or dashing cold water over the face.

The most interesting fits occur in the so-called "Jacksonian epilepsy." Here the fit always begins in one part of the body, perhaps the finger or wrist or face, and then spreads to other muscles. It may be limited to one limb, and the

patient be able to watch it during its whole course, or it may soon become general and, excepting for its onset, resemble one of true epilepsy. These cases are due to some local trouble in the brain, and one which can often be removed by operation.

The nurse can help greatly in the diagnosis of a case with "fits," since she sees the attacks oftener than does the doctor. She should note the patient's statement as to aura,—“how he knows when a fit is coming on,” for if the warning is a flash of light, a sound, an odor, a sensation in a certain part of the body, that may aid in determining in just what part of the cortex the fits begin. The muscles which first twitch should be noted. Perhaps it will be in one finger, or a corner of the mouth, or an eyelid, etc., but this shows the point where the “explosion” in the cortex began, and that is of great importance in treatment. During the convulsion the difference between a case of epilepsy and of hysteria is usually pretty apparent.

CHOREA.—“Acute chorea,” “St. Vitus’ dance,” is a disease the most prominent symptoms of which are constant, unconscious, irregular, purposeless muscular contractions. Whatever the child tries to do he does awkwardly, or fails to do, because he cannot control his movements. Even while he lies at rest, if his is a severe case, the involuntary movements of the face, limbs and trunk will continue.

This disease occurs especially in children. It seems to be an infection, for it so often occurs with acute rheumatism, acute tonsillitis, and acute endocarditis. Other cases follow a fright, or severe school work, etc. During the week before the attack the child is often irritable, wilful and emotional, in marked contrast to his usual disposition. Then begin the awkward, jerky movements. The child spills and drops things in his hands, makes grimaces. The movements may affect the hands only, “mild chorea,” or hands and face, or the entire body, “severe chorea.” The child may be unable to feed, or to dress himself, or even to talk. He is always weak. Slight mental symptoms are usually present, but sometimes he is really maniacal, “chorea insaniens.” The case lasts from eight to ten weeks, and for about six weeks more, during the convalescence, he should be under treatment to prevent sequelæ.

The treatment is, first of all, absolute rest in bed during the whole of the illness, and also during much of the convalescence. If possible the child should be separated from relatives, friends and especially playmates; from all except those absolutely necessary in his treatment. Good nursing consists in keeping the child quiet and amused without exciting it. The diet should be as full as possible. Of drugs, arsenic for the chorea, iron for the anæmia, and salts for the bowels are those most important.

It is important to prevent the children of nervous parents from acquiring the disease. It is especially important to protect them from an ambitious school teacher, also to curb their own ambitions. They should never enter any possible competition for a prize, etc. Children while in the prechoreic wilful stage are often injured by correction.

The movements of *habit spasm* or "tic," are often mistaken for chorea. Children with a tic frequently and at regular intervals make some spasmodic movement. It may be a quick nod of the head, a grimace, a twitch of the eyelid, etc., but it is always the same movement. The child sometimes accompanies the movement with an oath or other shocking word or expression.

CHRONIC CHOREA OF ADULTS, or "Huntington's chorea," is a totally different disease. It is inherited, the movements are slower than in the true chorea of children, and are more a series of writhing contortions. Speech is affected; there is gradually developing dementia. It is incurable.

MIGRAINE.—Migraine, or "sick headache," is an affection in which there are attacks of severe headache limited usually to one side of the head. Some persons have premonitory symptoms before the attacks, such as hallucinations of sight, visions of animals, persons, etc.; some become partially blind, some see a bright light, or colors. Some are mentally confused or depressed. The headache is of a particularly sharp character and spreads usually over half the head. Nausea and vomiting are very common symptoms. During the attack most patients are completely prostrated, and cannot endure any noise, light, etc.

These attacks last from one to three days. They recur frequently. They are brought on by excitement, by errors in diet, etc. They sometimes are much diminished in number

by a proper pair of glasses. As the attack comes on a brisk purge should be given, and the patient kept absolutely quiet in bed. Coffee may help diminish the nausea. Of drugs, a long list is recommended, including chloroform, caffeine, cannabis indica, antipyrine, antifebrin, phenacetin, ergot, etc., but the medicinal treatment is unsatisfactory.

VASOMOTOR DISORDERS.—The muscle coats of practically all the arteries of the body are under the control of nerves which thus determine the size of the vessels' lumen. These nerves are called "vasomotor nerves." Some, the "vasoconstrictors," make the vessel contract, that is, become a smaller tube; others, the "vasodilators," make them dilate. A common illustration of the influence of vasoconstrictors is the pallor of the face due to fright, while blushing is due to the vasodilators.

Raynaud's disease is a "vasomotor" disorder. In the mildest cases the person complains that exposure to cold, or an emotion, gives him "dead fingers," or "dead toes." The skin of these parts becomes white and cold, then, with the reaction, they become red and hot. The whole hand may be thus affected. In cases a little severer the fingers, toes, or ears, etc., become livid and swollen, stiff and very painful. This condition is popularly called "chilblains." In the severest cases the local cyanosis (lividity) is permanent, and the skin dies and sloughs off. Usually this affects only the tips of the fingers and toes, or of the ears, but the whole of the fingers or even the whole hand may slough off. An interesting feature of these cases is that the gangrene is usually symmetrical, that is, affects corresponding areas on both sides of the body.

ERYTHROMELALGIA, or "red neuralgia," is a similar condition to the above, and some think is the same. There is pain, with flushing and local fever of the feet or hands, worse when the parts hang down.

ANGIONEUROTIC OEDEMA.—By "oedema" is meant swelling due to the accumulation of fluid. It usually means that the subcutaneous tissue is swollen because the lymph of the affected part is much increased. In angioneurotic oedema the swellings are very local; the eyelid, lip, cheek, or hand, *e.g.*, may suddenly become huge. These attacks are usually accompanied by intestinal colic, pain, nausea, and sometimes by vomiting. If the swelling is of the tissue lining the larynx, rapid death may result.

CHAPTER X

DISEASES OF GLANDS

DISEASES OF THE THYROID GLAND

THE THYROID is the soft organ which can be felt on the windpipe just below the "Adam's apple" (larynx). It is very easily felt when it rises during the act of swallowing. This organ is a gland, and yet it has no duct, for it manufactures only an internal secretion (Fig. 98).

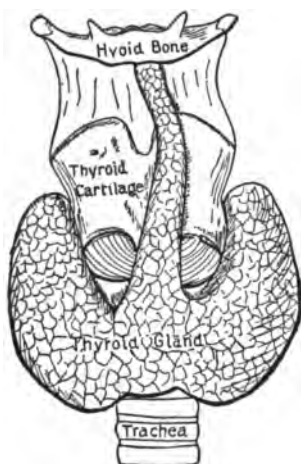


FIG. 98. The organs of the throat. The thyroid cartilage is the so-called "Adam's apple."

MYXEDEMA.—This substance which the thyroid supplies to the blood is one of the most interesting and best understood of all internal secretions. If the gland is removed or destroyed, the person begins to gain weight; the skin becomes dry and thick, and under it gathers a layer of mucilaginous tissue ("myxedema"); the hair dries and falls out; the pulse rate falls; the expression of the face becomes stolid, stupid and Esquimo-like; the person gets irritable, suspicious, and often quite "impossible;" the mind becomes duller and duller; and finally the patient may become a helpless idiot. This condition is named "myxedema" from condition of the skin.

One of the most brilliant of medical attainments is the discovery that we can supply this internal secretion by daily feeding the patient with the thyroid gland of animals, either fresh, or in the form of a dried powder (preparations spoken of as "thyroid extract"). The symptoms described above gradually disappear and, however distressing the condition has been, the patient in time may be entirely restored. We

also know that one of the chief ingredients of this internal secretion is iodine, and that some iodine preparations are almost as efficacious in treatment as the animal gland itself.

Some babies are born with the thyroid gland absent or practically so; in other children the gland is unable, in varying degrees, to do the work required of it; while in a third group the normal gland is practically destroyed during an attack of scarlet fever or some other acute disease. These children scarcely grow at all, or after a few years of fairly normal growth their development practically stops. A child of sixteen may, in size and appearance, resemble one of seven or eight years, etc. Mentally these children are very dull. Their faces are stupid, the mouth is open, the tongue hanging, the nose thick, the hair thin, the skin of the body very doughy. Saliva dribbles from the mouth. Such children are called "cretins." Their condition can be greatly improved by feeding them with thyroid extract. Of course the treatment must be continued for life, or the patient will relapse into this condition.

The condition of hypothyroidism (diminished thyroid function) is not rare. In a great many cases the gland seems fairly normal and yet it is unable to produce quite enough secretion for the needs of the body. A person in this state may have only one or two of the above symptoms in a very mild degree—for instance, a rather dry skin or falling out of the hair—and his real disease may be unsuspected. Thyroid extract in very small doses will improve the condition of which these patients complain, and it often, also, shows through their unexpected improvement in other ways how deficient they have been.

One rare condition may here be mentioned, since there is some evidence that it belongs in this myxedema group. "Dercum's disease," or ADIPOSIS DOLOROSA, is a disease characterized by a great increase of fat, which is painful to the touch, and which is usually deposited unequally over the body. The mental condition of these patients is not up to the average. A few cases have been given the therapeutic test—that is, did improve when fed with thyroid extract.

A GOITRE is a thyroid gland so large that it is really a tumor. Yet the new tissue which causes the swelling is in such a case not thyroid tissue. Sometimes the gland enlarges

as a whole, but more often only on one side. Goitres are rare in America, but they are very common, especially among women, in certain countries, particularly in Switzerland, Italy and parts of Asia. There must be some local cause for this trouble, as strangers settling in those regions may also become infected. The symptoms are due chiefly to the pressure of the tumor on the windpipe, and on the blood-vessels and nerves of the neck. Sometimes these patients become myxœdematous. The treatment is to cut out the most of the tumor.

EXOPHTHALMIC GOITRE, or "Graves's" or "Basedow's disease," is a condition in which there is a goitre,—that is, an enlarged thyroid gland,—but the gland is not nearly so large as in an ordinary case of goitre; in fact we may have to cut it out to be sure it is enlarged. But here there is an increase of thyroid tissue, and all the symptoms seem due to overproduction of the thyroid secretion. A few of the symptoms of this trouble may be produced by feeding a normal person with large doses of thyroid extract.

This disease often develops suddenly, following a fright, mental strain, etc.; many, indeed, think the local disease is due to a nervous trouble.

Exophthalmic goitre gives as important symptoms: a rapid heart (tachycardia), with the pulse between 100 and 160; prominent eyes (exophthalmos); a nervous disturbance in the movements of the eyes, so that, among other abnormalities, the whites of the sclera can be seen above the iris; a fine, rapid tremor of the fingers and hands; and a restless, apprehensive state of mind, best described as "crystallized fear." Patients with this disease perspire profusely, and often have gastro-intestinal disorders. The condition occurs especially in women.

As in myxœdema, so in exophthalmic goitre, there are many slight cases, the diagnosis of which is difficult. The only symptoms, indeed, may be "nervousness" or "heart disease."

The treatment of these cases is fairly satisfactory. Some of them recover without treatment, some recover during a "rest-cure;" others after X-ray treatment. But the best treatment for a severe case is to remove half the gland, on the ground that the trouble is too much internal secretion. The last stage of most patients that are left without treatment is shocking. They become very emaciated, intensely

nervous and delirious or even insane, and the heart "races" to death.

Behind and in the thyroid gland are from three to four other but smaller glands, about the size of a bean—the **PARATHYROIDS**. These also furnish only an internal secretion. When these are removed a fatal tetany results. By **TETANY** is meant a peculiar convulsion of the arms, which slowly become rigid, with all joints flexed.

DISEASES OF THE ADRENAL GLANDS

The adrenals also are glands with only an internal secretion. When for any reason this is absent, the condition known as "Addison's disease" results. Since this disease is usually due to tuberculosis, we have mentioned it under that heading (see page 280). Other diseases also, such as cancer, atrophy, inflammations, et al., may destroy these glands and produce practically the same clinical picture as tuberculosis (Fig. 73).

CHAPTER XI

CONSTITUTIONAL DISEASES

GOUT.—Gout is a "disturbance of metabolism," or a "nutritional disorder." These terms imply much more than they mean, for we know really very little about such troubles, although much about their results. For instance, diabetes mellitus is a disturbance of metabolism. The tiny body-cells cannot use sugar. Why, we do not know. The only wonder is that these wonderful little living creatures get along ("metabolize") so well as they do with the diet the blood supplies. In gout there is trouble, not with sugar, but with uric acid, of which there seems to be too much formed, and too little excreted; hence it collects in the blood. Whether or not uric acid causes the symptoms of gout is an open question. It is true that between acute attacks of gout the amount of uric acid in the urine is much diminished; that it is especially low just before one of these attacks; that during the attack it increases, perhaps to normal, but not much beyond; and that after an acute attack the patients do feel much better than before. In gout there are deposits of uric acid in and about the inflamed joints, and little masses called "tophi" in the ears and on the knuckles.

Gout is best described as the natural result of two evils of civilization—too much food and too little physical work. The best simile is that of an engine fed with too much coal, and hence with fire-box clogged. The disease may be acquired by the fortunate by too high living, too much meat, too much alcohol, especially the fermented liquors, and too little exercise; but the poor also suffer from it as the result of poor food, alcohol, poor hygiene, and the use of lead in their work (in the case of painters, etc.). Poor kidneys seem also to play a very important part. The worst forms of gout, however, are inherited, not acquired. The children of a gouty father may have typical arthritic attacks, but are more liable to have the troubles mentioned in the list on page 221.

The most important symptom of gout is acute inflammation of the joints. The joint first inflamed is usually the first joint of the big toe. The attack usually begins in the early morning, with agonizing pain in this joint, and later the other joints of the foot are affected. They become swollen, red, hot, and exquisitely sensitive, and the patient feels as if the foot were in a vise. There is fever. Towards morning the pain abates, may even disappear, and yet the joints may look just as inflamed as they did during the night. This is an important point in differentiating these cases from acute rheumatism. If we see a man walking with little pain on a joint which looks very inflamed, we are pretty sure he has gout. The next night the pain returns, and so on for from five to eight days, the severity gradually diminishing each time. These attacks usually recur, often with intervals of some months. Almost any joint may be involved, but the great toe rarely escapes.

In addition to joint troubles, there are many other symptoms which are as truly gouty. Gastro-intestinal attacks, with pain, vomiting, diarrhoea, and depression; cardiac attacks, with irregularity of the heart and its accompanying pain and shortness of breath; skin diseases, especially eczema; dyspepsia; arteriosclerosis and all its resulting troubles; headache, migraine; neuralgias, cramps; renal calculi; chronic bronchitis; and a host of eye troubles.

As a result of chronic gout, the joints, especially of the hands and feet, become considerably deformed. The deposits of uric acid may form on the knuckles knobs which may ulcerate through the skin (chalk stones). Patients with this disease almost always have arteriosclerosis and nephritis. They do not die of gout, but suddenly of some intercurrent infection (that is a terminal infection, causing pleurisy, pericarditis, meningitis or peritonitis, etc.) or of uræmia.

The gouty patient should be a total abstainer from alcohol, temperate as concerns food, and regular in his exercise in the open air. He should keep his skin in good condition by bathing daily. The diet should be varied, but very limited in quantity (yet not much less than any normal man should eat, for many men eat too much). Milk and eggs are especially good, and bread with much butter. Water should be drunk in great excess. "Diets" should on the whole be

avoided, since they are usually extreme in some point or other. It is better to starve for a few days and then begin with a very limited milk diet, taking next milk and eggs, and later a mixed diet of limited quantity.

During the acute attack the patient should be purged, the diet cut down to milk, the foot elevated and wrapped in hot compresses, and wine of colchicum given to relieve the pain and inflammation.

DIABETES MELLITUS.—Diabetes mellitus is a disturbance of metabolism. Glucose, the ordinary fuel of the body, for some unknown reason is not well used, accumulates in the blood, and is excreted by the kidneys. Although nephritis often develops as a complication, diabetes mellitus is not a disease of the kidneys, for their duty is to remove from the blood all glucose in excess of 0.2 per cent. Nor is it a disease of the blood. For some reason the tiny cells, the little living workshops of the body cannot use glucose well; yet the disease may not involve them, for they burn with ease other sugars, some, as levulose, very similar to glucose. But no other sugar can quite replace glucose, else the treatment of diabetes would be easy. We believe that the trouble is in the pancreas; that this organ furnishes the blood with an internal secretion which must be present in order that a cell may burn this sugar.

To find glucose in the urine once does not necessarily mean diabetes. We must find it during long periods of time. It is often present for a few days after injury to the head, and also in other conditions. Again, if one of us were to eat at one time 300 Gm. of sugar (glucose or even cane sugar) he would excrete some of it in the urine, for his body could not store up or use that large amount on so short notice. He would have "overstepped his limit." But if a person loses sugar in the urine after a 100 Gm. dose, we may suspect diabetes mellitus, for a normal person has a higher "limit" and greater "tolerance" than that. That is, our bodies have a "limit" to their ability to use glucose. They "tolerate" up to a certain extent. The limit of a mild diabetic is lower than normal, while the severe cases cannot tolerate even a few grammes, but will excrete every trace. A normal man has almost no limit for starches (bread, etc.), perhaps since it takes so long to digest them that the limit for glucose is never

reached. But the diabetic has a starch limit, and what this is in each case it is very important for the doctor to know. When we say that a diabetic's "tolerance" for bread is about 50 Gm., we mean that he can eat this amount at one time without glycosuria following; that after 60 Gm. a little sugar would appear in the urine. In the urine of severe cases there is always sugar, since they are never below their limit. They may take a diet entirely free from carbohydrates, they may even starve; but they are always burning a little of their own tissues, and this process furnishes some glucose. Cases of moderate diabetes can use a little of the carbohydrates with impunity, if these are taken in quantities within their limits; but let the patients overstep this, and sugar appears at once in the urine.

Some diabetics have inherited the disease; in some cases it dates from a severe mental strain, or a nervous shock, an injury, an acute infectious disease; in more cases no cause can be assigned. The anatomical cause is described on page 147.

The onset of the disease is often gradual, and the first symptom is usually an increase in the amount of urine. With this polyuria there is intense thirst. Much water is needed to excrete the sugar; this concentrates the blood, and the sensation of intense thirst is the result. If the amount of sugar excreted is diminished by dieting, the amount of urine at once diminishes, and the thirst lessens. Diabetics have voracious appetites; they have especially a terrible craving for sweets and starches. Their conscience is evidently affected, for to get these they will use all kinds of deceit. Sometimes they go on "carbohydrate sprees" and gorge themselves with ice cream, cake, and candy.

Unless the glycosuria is checked, emaciation soon begins, and then the patient tends to go steadily down hill. The "fat-diabetics" usually run a long, mild course, almost without symptoms. The "lean and hungry" diabetics are usually very severe cases; the younger the patient so much more severe the case.

The urine in mild cases may be normal in amount, but usually it runs from 3 to 4 litres; in severe cases from 4 to 10 litres are not rare; and in the severest it may reach 20 litres or more. Its color is pale greenish yellow, and its specific

gravity is high, from 1025 to 1045 or over. The amount of sugar varies with the diet, and if the patient eats all he wants it may reach a pound or more a day. A very severe case on a "carbohydrate free" diet voids about 50 Gm. daily. The milder cases are all sugar-free on a "carbohydrate free" diet, and remain so when carbohydrates are eaten in quantities below the patients' limits.

In speaking in the following pages of the urine we use the term "sugar-free" when no sugar is present, and "glycosuria" when it is present. Practically the only sugar in the urine in this disease is glucose or grape-sugar. The term carbohydrate is used because it covers all sugars and starches which, although quite different in the food, are soon practically all transformed by digestion to glucose. We eat very little glucose as such.

If even a normal person is put on a carbohydrate-free diet, acids will soon appear in the urine, and the most important of these is oxybutyric acid, an acid similar to that which gives the odor to rancid butter. These acids are present in much greater quantities in the urine of diabetics, and may finally cause coma and death, perhaps by their intoxication—"acid intoxication"—perhaps by using up the alkali of the body and thus causing an "alkali starvation."

Among the complications of diabetes are boils, carbuncles, severe itching of the skin, "pruritus," and gangrene of the fingers and toes, or even of a whole limb. Neuralgias, paralyzes, cataract of the eye, et al., are also common. Diabetics are very susceptible to infections (hence the boils, etc.), and die less often of diabetes than of a terminal infection, such as pneumonia, gangrene of the lung, pulmonary tuberculosis or blood-poisoning. The patients who die of diabetes itself die in "diabetic coma," and the first symptoms of this should always be carefully watched for. These may be a gradually increasing drowsiness, or headache, or nausea and vomiting. Soon begins the "air hunger;" the breaths are very deep and straining, although there is no impediment to respiration. The breath has a sweetish odor due to acetone. Once developed, coma is almost always rapidly fatal.

The prognosis in diabetes is always bad, so far as cure goes. Mild cases in adults may run for twenty years, but in children and young adults, the disease is usually rapidly fatal.

The treatment is chiefly general, for no drug is of use, except large doses of soda, when there is acid in the urine. If coma is feared, the soda should be given by mouth, by rectum, and intravenously, in the largest possible amounts, even 100 Gm. a day. This alkali is given to combat the acid intoxication.

Even in a mild case one must reorganize the patient's life. The disease is incurable, and the question is, how to prolong fair health as many years as possible. The patient should lead a quiet life, free from worry and hard work. Frequent bathing will keep his skin in good condition. He should avoid any excess, should guard against fatigue, against cold, etc.

We cannot here give the details of dieting these patients or copy the charts of the various permitted or forbidden foods, but we state the general rules which govern these diets. By "carbohydrate-free" diet we mean one in which the food we *give* contains no sugar or starch as such. But the food the body *uses* is never free from carbohydrates, for not only is sugar split off from the pure proteid molecule, but we are always burning up some of our tissues, and this process results in sugar. In reckoning the amount of carbohydrate in the food, these two sources must always be considered. The patient's tolerance of carbohydrates is the important factor to consider. The longer the glycosuria continues and the more sugar there is in the urine, the less becomes the tolerance. The patient handles the little he can use less and less well; that is, his limit gets gradually lower. On the other hand, the less the sugar excreted, in general terms the better becomes the tolerance, and even one sugar-free day (no sugar in the urine) will raise the limit perceptibly. Theoretically, therefore, a diet as free as possible from carbohydrates would be best. But such a diet is very hard on the body, for glucose is a most important food, and not even a normal man could stand doing without it long, much less the severe diabetics, who would soon be in fatal coma. We must always compromise. The problem is to avoid the dangers of progressive emaciation and acid intoxication on the one hand, and the injury to the patient's tolerance on the other; to keep the patient as nearly sugar-free as is possible and wise. Not only is each case a problem, but the problem in each case is

continually shifting as the intolerance shifts. In mild cases, where there is no sugar in the urine while the patient is on a carbohydrate-free diet, we add bread to the diet, increasing the amount ten grammes a day, until the first trace of sugar appears. Thus we get the limit. Suppose it is 80 grammes. We then allow this patient about half that amount every day. That is, each day he may have 40 Gm. of bread or its equivalent in any other carbohydrate. "Equivalent" does not mean equal in weight, but equal in sugar-value. These values are given in tables. For instance, the equivalent of 20 Gm. of wheat bread is 400 c.c. of milk, 70 Gm. of potatoes, etc. In this way a pleasant variety in diet is obtained. The limit should be tested from time to time, and the diet changed accordingly. One should remember that the various diabetic breads on the market all contain more or less of the carbohydrates, which the body turns into sugar, and in giving them one should make due allowance for this. Since levulose is better used than glucose, the attempt has been made to use that, but with only partial success.

Severer cases are sugar-free only on a carbohydrate-free diet, but this diet would in time kill them. If, however, the carbohydrates of their food are diminished daily, so that at the end of a week the diet is carbohydrate free, then often one "hunger day" of complete abstinence from food will so increase their tolerance that the next day a small amount of bread, which before this would have caused increased glycosuria, may be given without the urine's showing a trace of sugar. This amount may even be slowly increased without injury. They must have a little sugar in the diet. They should be carefully watched for signs of coma.

DIABETES INSIPIDUS.—This is a remarkable disorder, the chief symptom of which is polyuria—that is, the passage of large amounts of fairly normal urine. There is polyuria in diabetes mellitus also, but there it is due to the sugar which must be excreted; in chronic nephritis also, but there the cause is a very severe nephritis, and the urine is not normal; in hysteria, and after epileptic convulsions, etc., but it is very transitory; during and after acute fevers, but chiefly because we make the patient drink large quantities of water. But in diabetes insipidus the polyuria is chronic, often lasting many years. The kidneys, so far as we can now see, are nor-

mal, and the urine, apart from its diluteness, is also normal. This very rare disease occurs especially in young men. The cause may be primarily in the brain; some say that it is in the kidneys, and that these, able to excrete only a dilute urine, must in order to eliminate the solids, excrete large amounts of water. The result is a fearful thirst. The general health is little affected. The disease may begin at birth; in adults it sometimes comes on gradually, sometimes suddenly, after a fright or an injury. The amount of urine passed is often five or six litres, and in some cases over 20. It looks like water, and its specific gravity is from 1001 to 1005. It contains no abnormal substance, as sugar, albumin, etc. No treatment is of avail.

RICKETS.—Rickets is the name of a disease of infants, and is characterized by alterations in the bones at the time they are growing most actively, and by wasting of the entire body. It comes on insidiously during the first or second years of life. There is fever and the child becomes irritable and restless, especially at night, when he is always throwing off the bedclothes. He screams if touched, as if his whole body were sore. He sweats profusely, especially the head, so that the pillow is soaked. There are also digestive disturbances. The child will not learn to walk till late, and, if he has already learned, the rickets will put a stop to his walking. The mother fears he is paralyzed. There is great weakness of the muscles. The child may lose weight rapidly, or he may stay plump but flabby. The ends of the ribs and of the long bones swell, because of an overgrowth of soft bone. This "rickety rosary" of the ribs, and the large wrists and ankles persist through life. The chest becomes somewhat deformed (often "pigeon breast" is due to rickets). The child's head is large, his face small. The head looks square in shape, and the forehead is prominent. The skull often feels very thin, like a parchment, and the fontanelles do not close so early as is normal. Delayed teething is the rule. The bones of the arms become crooked if the child uses them much in creeping, those of the legs bend if he walks much. The legs may become bowed, or knock-kneed, but the most characteristic change is a forward arching of the shins, hence the name "saber legs." The abdomen is swollen ("pot belly"), partly because the liver and spleen are big, more because the bowels

are flatulent with gas. The child is usually peevish and irritable, sleeps poorly, has convulsions, and spasms of the throat, and is very susceptible to acute diseases. All grades of the disease occur, from the mildest, which are not noticed by the mother, to the severest, which produce misshapen dwarfs.

This disease is very common, especially, but not entirely, among the poor. Faulty diet is one cause. Sometimes the disease is caused by the mother's having children too near together, or nursing them while she is pregnant. In its slighter grades it occurs not rarely among the rich, especially those who use the various expensive proprietary infants' foods.

In treatment the diet is the first consideration. The child should have a healthy wet nurse, or be fed on properly diluted cow's milk. He should be kept perfectly quiet to prevent deformities; should, as much as possible, lie, warmly clad, in the sunlight and fresh air. Warm baths and sweet-oil rubs will greatly relieve the sensitiveness. Various medicines are a help, including phosphorus, iron, cod-liver oil, etc.

CHAPTER XII

THE SPECIFIC INFECTIOUS DISEASES

INTRODUCTION.—In addition to the world with which we are familiar, there is another world, vast, intensely interesting, and exceedingly important, which surrounds us on all sides, and which, although invisible to the ordinary eye, is of vast importance to the life of the plants and animals we know so well, and to our own life. We refer to the world of the minute plants and animals. While the activity of these tiny creatures is ever manifest, they themselves are not seen, unless vast numbers are crowded together, as for example, the dots and streaks on a crust of bread. To study the separate individuals we must use a microscope, which will magnify them. In fact, were a man magnified as much as are these tiny organisms, he would be over a mile tall. So tiny are they that the best milk on the market contains at least 600,000 to every teaspoonful; good milk over four millions; and the ordinary milk sold in cities, many times this number. Escape from this world of the micro-organisms we cannot, nor should we try, for the life of the world, including our own life, is possible only because these do play their part. In the great chain of existence the carnivorous animals use as their food the herbivora, and the herbivora, plants. Plants feed on the simplest gases—carbon dioxide and ammonia—on salts and water. But the origin of these gases and salts is the decomposed tissue of dead plants and animals. Suppose that the putrefying bacteria should suddenly cease their activity. Then each plant and animal when it dies would be embalmed by the sun, there would be no food for plants, and they would die. Next the herbivora and finally the carnivora, would all die, because this one link of the micro-organisms had dropped from the chain. But the organisms of decomposition and fermentation fill only one part of this world of tiny creatures. The nitrifying bacteria also are interesting. They “oxidize” the nitrogen of the air and the simple ammonia gases set free by

decomposition to nitrates,—salts which farmers at considerable expense add to the soil in fertilizers. These humble bacteria seem to do this easily, while many of our best chemists to-day are striving with poor success to rival them and oxidize the nitrogen of the air. Other micro-organisms have more aristocratic duties than these. The flavor of cheese, of butter, of tobacco, the results of the various “ripening” processes, the fermentation of beers and the rising of bread—all these are for the most part due to micro-organisms.

To go further. We are the “hosts” of myriads of these little creatures; they live on our skin and in our intestine, and it is doubtful if we could well get along without them.

In this vast host of micro-organisms of tiny plants and animals, there are many species, races, and varieties, which an expert can distinguish as correctly as can you in a flower garden pick out the roses from the lilies, pansies, etc. Not only that, but of each of these species one can distinguish varieties, just as the florist distinguishes the tea from moss roses, and among the tea roses distinguishes the Jacks, Richmond Reds, etc. Among all of these there are a few varieties which in most particulars are very like their “cousins,” but which are decidedly poisonous to us and which man should try to exterminate, just as he tries to kill off among the reptiles the rattlesnakes. They are the so-called “virulent parasites,” micro-organisms which live at the expense of a host and injure him. They are comparatively few in number. Many are well studied and quite thoroughly understood, and practical methods for their extermination have been devised.

Some of these virulent parasites are true parasites,—that is, they can live only with a host. Others are only partially parasites; they can live independently,—that is, they can “support themselves” when no host is at hand. But we wish to emphasize the following point: just as sharp a line should be drawn between these pathogenic micro-organisms and the others as is drawn between a rattlesnake and a black snake, or, better, between an edible mushroom and a toadstool. The one we should try to exterminate; the others we should use.

Of this great host of micro-organisms, some are animals, some plants, and some so like both that it is hard to say which they are. In each of these three groups there are a few pathogenic or disease-producing, organisms. Of the animals we

shall speak later (page 325). The great intermediate group which stands between plants and animals, includes the great host of bacteria, and we know more about these than other germs, because they are so easily studied. The group of the pathogenic animal micro-organisms is an ever increasing and vastly important group, as it is more than probable that some of our worst diseases are due to them. They cannot yet be studied as easily as bacteria, since we cannot now grow them in our laboratories, keeping them and their descendants alive for years, as in the case of most bacteria.

Pathogenic micro-organisms are popularly called "germs." Some persons still speak of the "germ theory of disease," as if there were still ground for a difference of opinion as to whether germs really cause disease or not. Not many diseases are caused surely by germs; concerning many others there is still doubt; but there are a few diseases which we are just as sure are caused by certain germs, as we are that the little red, itching spots on our skin were caused by the mosquito which we killed in the act of biting; or that those painful papules were caused by the hornets which swarmed from the nest we disturbed unwittingly. These diseases are few in number, but in every case of each of these diseases a certain germ can always be found by any one man who knows when and how to look; this germ and many generations of its descendants can be grown in glass tubes in our laboratories; and it or its descendants will reproduce the original disease in a man, or in certain animals, into which it is injected, accidentally or purposely (Koch's laws). Of course all animals, as man, dog, rabbit, etc., are not susceptible to the same germs.

Some diseases are due always to one and the same parasite. Among those caused by bacteria are acute lobar pneumonia, typhoid fever, epidemic cerebrospinal meningitis, cholera, lockjaw, glanders, tuberculosis, anthrax, influenza, erysipelas, diphtheria, and a few others. Among those caused by animals are malaria, "sleepy sickness," amœbic dysentery, lues, and a few tropical diseases. These are called "specific diseases," and the parasite of each is its "specific organism." Some diseases can be caused by any one of several germs, as a boil, a carbuncle, rheumatism, acute endocarditis, empyema, peritonitis. Inflammations with pus production are caused by a group of germs called "pyogenic" or "pus-producing" organisms.

The yeasts and moulds very often cause superficial inflammations, as thrush of babies' mouths, and trivial infections of the nose, mouth, ears, etc.; some seem able to cause lung disease, or at least to aid pathogenic bacteria to do so, while some cause bad skin diseases.

In the case of these "specific diseases" the evidence for the specific organism is so strong that the only rational way to doubt the pathogenic action of these micro-organisms is to doubt the existence of the disease they cause.

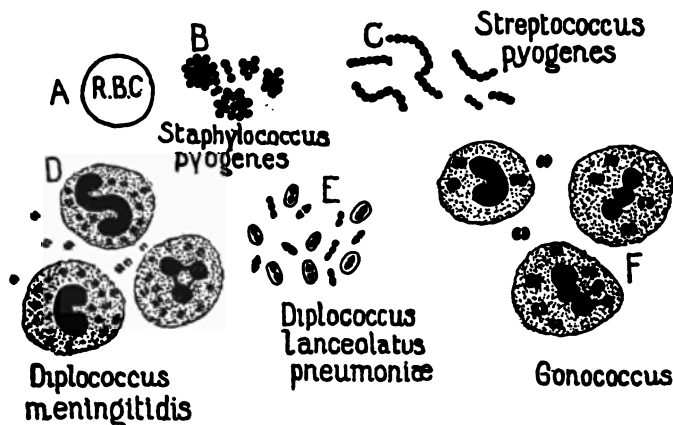


FIG. 99.—Vegetable parasites: Bacteria; Cocci. (All drawn to same scale, magnified 1000 times.) A—A red blood-corpuscle, drawn to the same scale for comparison of size. B—*Staphylococcus pyogenes* (aureus or albus). The cause of boils, pimples, abscesses, etc. C—*Streptococcus pyogenes*. The cause of erysipelas and "blood poisoning." D—*Diplococcus meningitidis*, or the "meningococcus." The cause of epidemic cerebrospinal meningitis. E—*Diplococcus lanceolatus*. The cause of pneumonia. G—The *Gonococcus*.

The simple presence of these germs does not cause disease. It is the poisons, called "toxins," which they produce that make the trouble. Each germ produces one or more toxins, which are peculiar to it, and which can be produced by no other germ. These are called "specific toxins." Some of these can be obtained separate from the germ, such as that of the diphtheria bacillus; some toxins cannot be separated from the body of the bacillus, as that of the pneumonia germ. It is safe to say that, if the toxin of each germ could be obtained from its organism, the cures of the diseases it causes would be as simple and satisfactory as is now that of diphtheria.

Pathogenic germs of all sorts are constantly gaining access to our bodies, but our body fluids have natural powers of destroying them. If the germ succeeds in influencing us in any way, or in causing any symptoms, we are said to have an

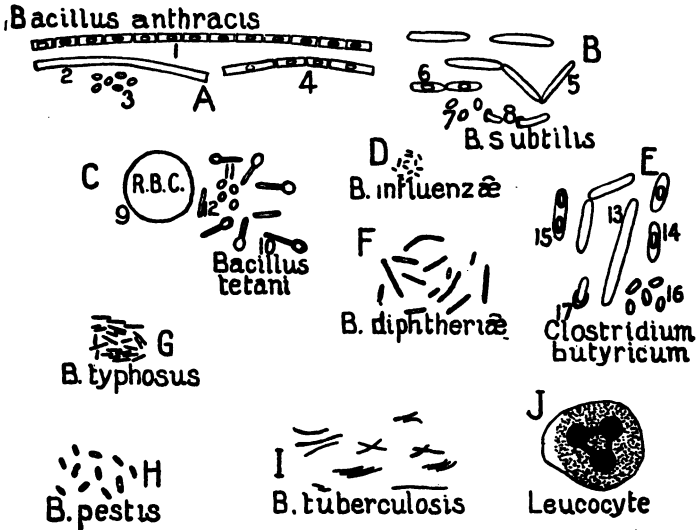


FIG. 100.—Vegetable parasites: Bacteria; Bacilli. Important disease-producing and harmless bacilli. (All drawn to same scale, magnified 1000 times.) A—*Bacillus anthracis*. A very dangerous bacillus, the cause of "anthrax," "malignant pustule," "wool-sorter's disease," etc. 1, a chain of bacilli, each containing a spore; 2, one long bacillus; 3, free spores; 4, a short chain. B—*Bacillus subtilis*. A harmless ubiquitous bacillus, also called "hay bacillus." 5, a short chain of bacilli without spores; 6, two bacilli containing spores; 7, free spores; 8, two spores "sprouting," that is, developing into bacilli. Note that this bacilli "sprouts" from the side of the spore. C—*Bacillus tetani*, or the bacillus which causes "lockjaw." 9, a red blood-corpuscle drawn to the same scale, introduced for comparison of sizes; 10, the "drumstick-shaped" bacilli containing spores; 12, a bacillus without a spore on the left, free spores on the right of "12." D—*Bacillus influenzae*, the cause of "la grippe." This germ produces no spores. E—*Clostridium butyricum*. A large harmless bacillus. 13, bacilli without spores; 14, bacilli containing spores; 15, a bacillus containing two spores; 16, free spores; 17, a spore "sprouting." Note that this bacilli develops from the end of the spore. F—*Bacillus diptheriae*. There are no spores formed by this germ. The dots are not spores, but indicate irregularity in staining ("beading"). G—*Bacillus typhosus*, the cause of typhoid fever. The same picture will do for *Bacillus coli communis*, *Bacillus dysenteriae*, etc. No spores are produced. H—*Bacillus pestis*, the cause of bubonic plague. The dots do not indicate spores but irregularities in staining ("polar staining"). No spores are produced. I—*Bacillus tuberculosis*, the cause of consumption. No spores are produced. J—A leucocyte drawn to the same scale for purpose of comparison of size.

"infection." When a germ can settle anywhere on the surface of the body, or gains access to our blood and is carried about and lodges in any organ, and multiplies there,—we have where it settles a "local" infection. If the germ lives in the blood and multiplies there, the condition is called a "septicæmia."

If the germ of a septicæmia is "pyogenic" and settles in any one of various organs, causing an abscess at the points where it settles, the condition is called a "pyæmia." In the case of those diseases surely caused by specific germs, the tendency now is not to make a diagnosis of this disease until the germ, or unquestionable evidence of its presence, is found.

In nearly all acute fevers caused by specific germs, the organism must enter the body at some one or more points. These are called "portals of entry." Some enter through the lungs, others through the intestines, the tonsils, the kidneys, etc. It is then carried throughout the body by the blood. Each germ has a favorite organ in which it is especially apt to settle and cause disease. The typhoid germs select especially the intestine; some, as the tubercle bacillus, select the lung; some the brain, some the heart valves, etc. Wherever a germ settles it produces its "local lesion," as distinguished from the diffuse lesions which its toxins may produce. If the local lesion is a centre for the growth and spread of the germ, it is a "focus."

The invasion of the hostile germ is the "infection." The symptoms may be limited to the local lesion, as the pain of a pimple, or be general, as the fever, headache, and malaise accompanying a carbuncle. In the latter case, of course, there are severe local symptoms also. Some infections produce no local lesions at all. This is true of some severe cases of blood-poisoning, or septicæmia. If during one disease the organism of another also invades the patient, the second invasion is called a "secondary infection." These are common, as the first disease robs the body of much of its resisting power. When this secondary infection causes death, it is called also a "terminal infection." Terminal infections are responsible for most deaths. Few persons "die of the disease they have," for their chronic disease, which is causing a slow, lingering death, is suddenly robbed of its victim by an acute terminal infection, especially pneumonia, which in a few hours or days proves fatal.

There are certain symptoms common to most infections, such as fever, headache, and general malaise. But, just as the symptoms produced by various drugs—opium, strychnine, etc., differ much, so those caused by various toxins (which also are "drugs") vary much. The toxin of typhoid fever causes

severe headache and stupefies the brain; patients with streptococcus infections have, usually, very clear alert minds; those with tetanus are abnormally alert and suffer from even a slight noise. These poisons have a markedly selective action. They all circulate in the blood, but some poison brain cells (tetanus); some, nerve endings in certain muscles (diphtheria); etc.

After the germ causing a fever enters the body it lies latent for a variable number of days. During this time, which

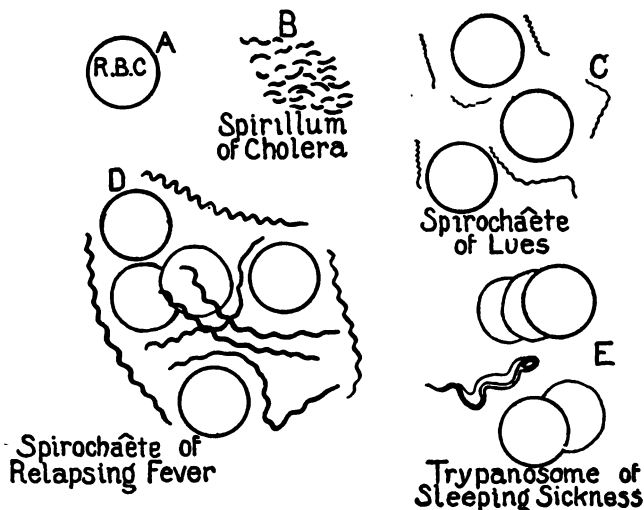


FIG. 101. Pathogenic organisms. A, a red blood-cell for comparison of size. B, the organism of Asiatic cholera. C, the organism of lues or syphilis. D, the organism of relapsing fever. E, the organism of sleeping sickness.

is called "the period of incubation," the germ is probably multiplying but is producing no symptoms whatever. With the first symptom, begins the period of invasion, which lasts until the disease is well established.

BACTERIA.—The bacteria are tiny organisms which are neither plants nor animals, but which resemble the plants more than they resemble the animals. The tiny round ones are called "cocci" (See Fig. 99). They need not be perfectly round; some are flattened together in pairs—"diplococci;" some are a little oval, or lance-shaped, as *Diplococcus lanceolatus*. Some are always in groups,—the

staphylococci; some are in chains,—the streptococci. Those that look like a stick are called bacilli (Fig. 100); those that are curved, spirilla (Fig. 101). Some are long threads which branch,—the “streptothricæ.”

In this chapter we shall treat of the specific bacteria causing specific diseases.

Bacillus Typhosus.—Nurses should know a great deal about this bacillus, as the treatment of TYPHOID FEVER is for the most part a nursing problem, also because many nurses contract this disease from their patients, and especially because they can aid much in the popular education which in time will entirely eradicate typhoid fever.

America suffers terribly from her negligence. Typhoid fever holds fourth place in her mortality list, killing every year over 35,000 individuals, chiefly young persons under 35 years, while over 400,000 others yearly suffer from this long, serious illness. In our Spanish-American war, one soldier in every five caught this disease. There were 20,730 cases and 1580 deaths; thousands it left physical wrecks for life, and this chiefly in the regiments which did not go to the front; and yet all this suffering and death were needless. In the Japanese army during seven months, including the summer and fall, there were, in an army almost five times as large as ours, but 133 cases. During the Chinese-Japanese war of a few years ago, the Japanese suffered from typhoid fever and learned their lesson. In American cities, and even more in our country regions, it still runs riot; while in some parts of Germany, where formerly it was common, it is now comparatively rare.

Bacillus typhosus (Fig. 100) is really a “distant cousin” of the colon bacillus, an ordinary saprophyte, which is always present in our intestines, but which seldom causes disease. Yet the typhoid bacillus is not the colon bacillus any more than the deadly toadstool, *Amonita*, is the same as the highly prized edible mushroom. Between these two organisms are a multitude of “cousins” that do cause disease. Some resemble more strongly the typhoid bacillus and are called “paratyphoid bacilli;” others resemble the colon, and are called “paracolon bacilli.” While *Bacillus typhosus* is a parasite, it may live for some time outside the body. This is important to know, as stools and urine of many persons who have had

typhoid fever, are at times, perhaps for forty years, swarming with this germ, and only the scientist knows how much of other persons' stools and urine we eat and drink daily. Deficient sewers are a great danger in cities, and cesspools find ready underground connection with wells. Often a single case of typhoid fever near the head waters of a city supply has infected a whole city. Contaminated water supply is a very great danger, yet here the harmless saprophytic bacteria, which so disgust us, are our safeguard, for because of them typhoid bacilli can live in ordinary water but about three days, while in pure water they can live for a week. Sometimes oysters seem to preserve alive these typhoid bacilli, but this danger is not great. Frozen in ice they may live for a long time. We little know how much fecal matter is carried on the feet of flies, which will impartially feed on a manure pile and then in a few minutes light on our plate, or how much filth there is in the dust that blows into our houses; and *Bacillus typhosus* can live in street dust for even 70 days. We "do not wish to know" how dirty are many who handle our green vegetables, or how filthy are the farm hands on some little out-of-the-way farm that sells a few quarts of milk to the agent who collects from a whole region milk for some distant city. One case of fever in the family of a dairyman near Baltimore recently caused an epidemic of over two hundred cases. Unfortunately, *Bacillus typhosus* does not change the appearance of the milk, cream, or butter, as do the harmless saprophytes, which sour it, but which do us no harm. The danger with this germ does not end with the disease. Some cases of typhoid fever get perfectly well, and yet the germ lives on in the urinary bladder or gall-bladder and multiplies there without bothering them. Years after the fever these persons' urine or stools may be simply alive with these germs. Such persons are called "disease spreaders." The germ causes them no trouble, but may give typhoid to someone else. If a man who has never had typhoid fever swallows a few germs it is not certain that he will become ill. Much depends on the "health" of the bacilli, for many of them are half dead; much depends also on the "health" of the person, for his natural protective powers may be sufficient to prevent the disease. When the "soil" is ready, then a few living bacilli are enough.

Bacillus typhosus enters our bodies by the mouth, through the medium of "fingers, flies, or food." It penetrates the walls of the gastro-intestinal tract. There it multiplies rapidly, is carried around the body in the blood in vast numbers, and very soon settles in some organs, where it grows. It now disappears from the circulating blood—that is, the septicæmia ceases, and a local infection begins. The most common seat of this local infection, and possibly a constant one, is the intestine. Just as we have tonsils in our throat, so we

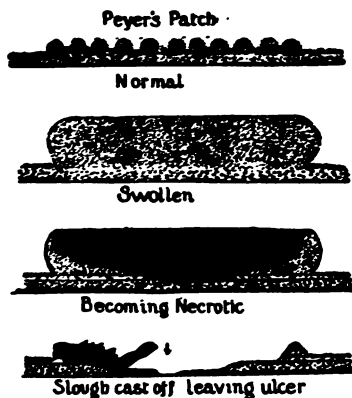


FIG. 102. The Characteristic Lesion of typhoid fever. (Magnified slightly.) In this fever the Peyer's patches first swell, then this swollen tissue dies and is cast off as a slough, leaving an ulcer. The arrow indicates a point where the bowel wall may perforate and peritonitis result.

have many of them in our bowels, some tiny, some large, but made of the same "lymphoid tissue," and perhaps with just the same function (whatever that may be), as the tonsils in our mouth. These large "tonsils of the bowels" are most numerous in the lower two feet of the small intestine, just above the ileo-cæcal valve. Here they form in the bowel wall broad, thin, flat sheets, which are called "Peyer's patches." There are also, all along the walls of both bowels, many minute tonsils, not so large as the head of a pin, called "solitary follicles."

These masses of tonsillar tissue

seem especially affected in typhoid fever. The Peyer's patches (Fig. 102) instead of remaining about as thick as writing paper, swell and in eight or ten days are even an eighth of an inch or more thick. The solitary follicles and the other lymphoid structures, especially the lymph-glands in the abdomen, also swell and may form palpable masses. Then the blood-vessels supplying the Peyer's patches become thrombosed, and the whole swollen patch dies, possibly because so exposed to injury from the intestinal contents. The solitary follicles suffer in the same way, but are so tiny that it matters little. The dead tissue of the patches sloughs out, leaving in the bowel wall a deep hole which may almost or quite perforate the entire wall and cause peritonitis.

After all the dead matter has sloughed away and the ulcer is "clean," healing begins, and soon there is no trace left of these deep ulcers.

It is of interest that the paratyphoid and paracolon bacilli, near cousins of *Bacillus typhosus*, cause a fever which is the same as typhoid, except that the intestinal lesions are absent, or are atypical.

Typhoid fever is preceded by a few days or even weeks of "prodromal symptoms." The patient feels badly, has some headache, some backache, perhaps a nose-bleed. He is usually constipated, and the tongue is thickly coated. We suppose that the few germs swallowed are multiplying in the body until numerous enough to start the fever. Soon, it is hard to say when, the temperature begins to rise, and the patient feels so weak and miserable that he goes to bed. The temperature rises slowly and in from three to seven days reaches its highest point, usually from 104° to 106° F. There it usually stays for a week or more, then it becomes irregular, as it drops each morning, but is high in the afternoon, so that the curve looks like a "snake fence." This continues for a week or more. Finally the morning temperature reaches normal. Then the afternoon record daily gets lower, and at the end of a week perhaps that also is normal. The duration of the fever is very variable. Some patients are well in two weeks, the majority in from four to six weeks, while in other cases the disease hangs on for even eight weeks or more. (We refer above to those cases without relapse.)

After the fever has gone, convalescence begins. The patient is at first thin and weak, but he slowly returns to good health or to even better health than he formerly had.

The pulse in a strong man is usually remarkably slow, considering the height of the fever. It is often from about 80 to 90. It is "soft" and one can feel the dirotic wave so distinctly that it feels like a second beat. The heart is often weakened by the fever, and this is not infrequently the cause of death. During convalescence the pulse-rate is often very slow.

For the first few days of the fever the headache is very severe, sometimes terrible. There is pain in the back and limbs also. As a rule, in about ten days these disappear and the patient, now dull, stupid, "typhoidal," does not suffer

at all. Then delirium may begin and may be a serious feature. In very toxic cases the patient lies quiet, the mouth open, the tongue dry. It is very hard to rouse him. There is considerable tremor of the arms when he tries to move them; he may pick at the bedclothes (*subsultus tendinum*), which is a very bad sign. At first he may vomit a little and have diarrhoea or constipation, but vomiting soon ceases. Diarrhoea is always an unfortunate condition, since the patient who remains constipated usually does better. Sometimes the abdomen becomes distended with gas.

At the end of the first week little rose-red spots, which disappear entirely on pressure, appear on the body, especially over the abdomen, the "rose spots" which are of great value in diagnosis. They are due to a colony of typhoid bacilli, which are brought by the blood and which settle here.

The spleen soon gets large. The leucocytes decrease rather than increase in number, as is the rule in infections, and usually are below 5000 (normal 7000 to 10,000), sometimes even 1500, per cubic millimetre.

Typhoid fever is a self-limiting disease,—that is, the body cures itself by forming a sufficiency of immunity substances. (See page 288.) When this is done, recovery follows. Once well, the patient is truly well, for second attacks of typhoid fever are exceedingly rare. But it takes a long time for the body to get sufficiently immune; there is scarcely an acute disease in which relapses are so common. When a relapse takes place, the temperature after reaching normal rises again, and the whole course is gone over a second time. When the relapse begins before the initial attack has subsided, it is called an "intercurrent relapse." There may be four or five such relapses before the patient is well, and hence the disease may last even six months. The relapses are certainly reinfections; perhaps new patches become swollen. There is no way of foreseeing a relapse. After the temperature has reached normal, it may rise again irregularly for several days, causing considerable anxiety. Some of the post-febrile rises are due to constipation; some, to an increase in diet; many, to the excitement of seeing friends. If, however, the rise is progressive for two or three days and the general symptoms return, it is probable that a relapse has taken place. Sometimes, and especially with children, the temperature will not for a long

time reach normal. As a rule, after the fever, the temperature is for several days considerably below normal—"hypothermia," and the pulse very slow, even between forty and fifty a minute. These are by some considered good signs; to others they are symptoms of starvation. (See page 247.)

Hemorrhages occur in at least 5 per cent. of all cases, and usually during the third week. They cause about 10 per cent. of the deaths. Evidently an ulcerating Peyer's patch, when the slough peels off, contains a small artery not yet tightly thrombosed. Some patients have repeatedly small bloody stools, others have a sudden, fatal hemorrhage. These usually come without warning, although the severe hemorrhages cause a drop in the temperature, the mind becomes clearer, and sometimes there is an indefinable feeling of anxiety. Fairly large hemorrhages show beautifully why a few years ago bleeding was so favorite a treatment for fever. In a few minutes after the venesection, a very ill, delirious patient with high temperature is clear-minded, his temperature falls to normal, the pulse scarcely rises, and all looks better. But the temperature soon returns to its former point, and the fever continues. The only difference is that the patient is weaker because of the loss of blood. In some conditions, as uremia, bleeding is now done to remove as much poison as possible. Unfortunately, in the case of bowel hemorrhages, we cannot stop them when we wish.

Perforation is the most dreaded complication of typhoid fever, and the cause of death in almost a third of the fatal cases. When the slough peels off, the ulcers usually have a very thin base, sometimes as thin as tissue paper, but in about 5 per cent. of the cases even this gives way, and the intestinal contents pour out into the abdominal cavity, at once producing peritonitis, which without operation is almost always fatal. In the very few cases that do recover, there is in the abdomen an abscess which later may require operation. A perforation occurs especially during the third week, although it may any time (as we reckon the days), and since due to almost the same cause as hemorrhage, occurs very often with this.

Of all the symptoms during typhoid fever, there are none so important for the nurse to know as those of perforation. Every hour, every minute, after the first symptom, is of the

greatest importance, for early operation is the most successful. Hence the nurse should know the danger signals and at once report them to the doctor in charge. The patient's life is in her hands, and it is better to give many false alarms than to fail to give one at an opportune time. Our rule is that if a typhoid patient voluntarily complains of abdominal pain, or hiccoughs or vomits, this shall be reported at once to the doctor. This initial abdominal pain is peculiar. It is sharp and sudden; the patient usually cries out with it; it is different from any pain the patient has had before; it lasts but a few seconds, then stops, and the patient in a few minutes may be sound asleep. This last point puts the nurse off her guard, for she can hardly believe that so momentary a pain, which ceases so soon and is followed by sound sleep is very important. Many cases have abdominal pain during the whole course of the fever. Seldom does this peculiar, sudden, sharp pain fail. It is one of the most reliable of the early symptoms. There are other early symptoms which may attract attention; a rise in temperature followed by a sharp fall, but this is not uncommon in typhoid fever; a slight increase in frequency of respiration, which is more important; sudden vomiting not immediately following nourishment; hiccough, which is rare, but which, when it does occur, even though heard but a few times, is very suspicious indeed; and pain on urination.

Soon after the perforation all symptoms may cease, and we may congratulate ourselves that the alarm was false; and yet even then a peritonitis may be rapidly spreading. If left alone the abdomen becomes more and more distended; the patient may vomit occasionally for a few hours or several days; he looks more and more ill and gradually becomes weaker and weaker until death.

The doctor will see whether the abdomen is sore at any point on pressure, whether leucocytes have risen, whether there is free fluid, or gas, in the abdomen, and will make various other tests; but the nurse should remember that the earlier he sees the case the easier will be the diagnosis. If he arrives even a few hours late, a diagnosis may be impossible without watching a long time, and may then be very doubtful. The general peritonitis in typhoid fever, unlike that of appendicitis, has very few symptoms, probably because the typhoid poison is a narcotic which masks the symptoms, much as

morphia would mask those of appendicitis. With early operation at least a third of the cases can be saved.

The typhoid bacillus enters the body through the gastrointestinal canal, and produces here the most marked local lesions. Hence the most prominent symptoms of typhoid fever are intestinal, but it is a general disease. During the first week the blood is simply alive with these bacilli, which may settle in almost any organ, producing symptoms referable to that organ. They may not attack the intestine at all, or attack it so slightly that there are no intestinal symptoms to suggest typhoid fever. Since other organs are sometimes affected early in the course of the disease, the first diagnosis is often a very wrong one. Some cases begin with symptoms of a severe cerebrospinal meningitis,—with convulsions even. Some begin with acute pleurisy; others with severe acute bronchitis, others with a pneumonia, while still others have at onset an acute nephritis. It is only after these conditions clear up somewhat, leaving the patient still with a high fever, that the true condition is suspected.

During the course of typhoid fever, complications are very common. Some are due to a fresh localization of the typhoid bacillus, which attacks a new organ, others to a secondary infection by another organism, which now has a chance, as the typhoid infection has so reduced the resisting power of the patient. Examples of the latter complications are the terrible bed-sores, the crops of boils, the parotitis, mouth infections, abscess of the liver, etc. *Bacillus typhosus* may locate in the pleural cavity and cause empyema. It may cause "periostitis" (infection of the outer membrane of the bones), venous thrombosis, abscess of the kidneys, ulcers of the larynx, acute endocarditis, myocarditis, pericarditis, pleurisy, acute meningitis, acute nephritis, acute cholecystitis, pneumonia, and infections of eyes and ears. The bed-sores are sometimes terrible. They occur with the greatest frequency if the patient is not kept clean and dry, or if too long pressure on the back, sacrum, buttocks, heels, etc., is not prevented by pillows and rings and by frequently turning the patient. The skin should be carefully examined every day, and if any suspicious spot is noted, this should be dressed, and protected from all pressure by a ring. And yet in some of the best nursed cases, bad bed-sores cannot be prevented,

as the protective powers of the patients are so reduced that they cannot resist the common ubiquitous organisms. The mouth should be rinsed out and swabbed out repeatedly every day to prevent mouth infection.

The nurse should watch any swelling of the legs, for venous thrombosis, especially of the left femoral vein, is very common. If it occurs, an ice-bag is kept over the vein, and the patient is moved with great care.

The diagnosis of typhoid fever is for the doctor to make, and yet a few points may help the nurse. That a man feels practically well and keeps on with his work does not mean that he may not then be having typhoid fever, for these "ambulatory" cases are frequent enough. Nor, is the fact that the fever suddenly ends in fourteen days or less, much of an argument against the diagnosis of typhoid fever, for some cases do this. Nor is this an argument that the last medicine taken "cut the disease short." Nor is the fact that the patient has many severe chills and sweats any justification for the diagnosis "malaria" or "typho-malaria" or "malarial-typhoid," for a true combination of these two diseases is rare, and the feeling of false security and the laxity in watchfulness, resulting from these diagnoses, are dangerous.

"Typhoid," "typhoidal," "bilious fever," "continued fever," "nervous fever," "low fever," etc., are terms which antedate the discovery of germs. They served, and still serve, a very good purpose. But the tendency now is to limit the term "typhoid fever" strictly to those cases in which we can prove the presence of *Bacillus typhosus*, no matter what symptoms the case may give. For many cases which look exactly like typhoid are not typhoid, and true typhoid presents itself in a great variety of guises. It is true that if a case has a typical temperature and a typical pulse chart, a history of typical prodromals and of typical initial symptoms, typical rose spots, a low leucocyte count and a spleen easily felt, we are satisfied to make the diagnosis of typhoid fever, but perfectly "typical" text-book cases are about as exceptional as are living "composite pictures." "Typhoidal" is a descriptive term. Typhoidal fever means a long fever which makes the patient dull and stupid. But a severe septicæmia or toxæmia due to almost any germ, but especially the malaria plasmodium and the tubercle bacillus, can make the patient

almost exactly resemble a patient with typhoid fever. "Continued fever" is the term applied to cases with a regular fever which lasts three or more days, the exact cause of which is uncertain. Among these cases are certainly many of typhoid fever, very many of tuberculosis, some of malaria, a few of pneumonia without chest symptoms, many of abscess (and small perirectal abscesses can closely simulate typhoid fever). But a more exact diagnosis than continued fever we do not make unless sure. The certain diagnosis is now made in the laboratory rather than at the bedside. The surest way to make a diagnosis during the first week of the illness is to find the bacillus itself. This is done by removing a little blood, with the greatest precautions against contamination (which means careful work by the nurse in cleaning the arm), and seeing whether we can grow a germ from this. During the first week one can, in true typhoid fever, get the germ in practically every case.

After the first week we may find it in the blood, in the urine, in the stools, or in the rose spots, but the easiest method of diagnosis then is the Widal test. As before stated, germs produce disease by means of the poisons which they produce. Each germ of disease produces a poison peculiar to itself. As soon as this poisoning begins, the body begins to manufacture antidotes, called antitoxins, and these are each an antidote for but one poison. When these have been provided in sufficient numbers, the patient begins to get well. If we can, therefore, prove the presence of an antidote, or an antitoxin, we are as sure of our diagnosis as if we had found the toxin or even the germ. There is a great variety of these antitoxins formed in each case of typhoid fever, and the one we use in the Widal test is the one which "clumps" *Bacillus typhosus*. A few drops of blood are allowed to clot, and the clear serum is taken. One drop of a fresh culture of a germ we know to be *Bacillus typhosus* is also taken. If these two drops are mixed, probably in nearly every case every bacillus will soon be killed, for our blood is provided with substances which protect us against the host of germs which frequently gain entrance to our bodies. But if one-fiftieth of one drop influences these germs much within one hour, stops every one from swimming and makes all gather in clusters, then we can be sure that the person is manufacturing a special anti-

toxin for that particular germ, for this amount of the serum of any person, well or ill with anything but typhoid fever, could not do it. Sometimes $\frac{1}{10}$ of a drop is enough, but the standard used is "a dilution of $\frac{1}{10}$ causing in one hour cessation of motility and agglutination." By this method we can distinguish between very nearly related organisms, and in any doubtful case of fever, if the typhoid bacillus is not agglutinated, the various paratyphoid and paracolonic organisms are also tried. The antitoxin, or protective substance, may be a mixture of several substances, for three changes may occur. The bacilli, which are active swimmers, stop moving entirely; the separate bacilli should gather into clumps—that is, should "agglutinate;" the bacilli may disintegrate and disappear—"bacteriolysis"—but this is not essential.

The convalescence of typhoid fever is long and tedious; it may even take many months. The disease has various sequelæ: among these are, "neurasthenia" of a very aggravated sort, for the shock given to the nervous system by typhoid fever is very severe, hence the name "nervous fever;" chronic arthritis of the spine, which explains some of the "typhoid spine" (painful stiff backs which may last months and greatly worry the patient); chronic neuritis, which makes the person lame for a long time; and the formation of gallstones.

The *treatment* of typhoid fever is chiefly a problem of nursing. One should remember that without any medicinal treatment, without cold baths, with only ordinary home nursing, at least 85 per cent. of the cases will get well; that with all that science can suggest as to the method of treatment and of nursing, the mortality is reduced from 15 to about 8 per cent.; and that our struggle is now to save these 7 per cent., perhaps later, the remaining 8 per cent. The nurse keeps the patient in so good condition that he can battle to the best advantage with the infection; she is on the watch for any symptoms which may require active interference; and, lastly, she tries to prevent the patient's family and the community from catching the disease.

The diet is a matter of greatest importance. There are three views in regard to it. Some believe that a milk and albumin diet is enough. Every two hours when awake the

patient takes either four ounces of milk diluted with two of lime water, or the white of one egg in lemonade. If the milk is not borne well (as shown by curds in the stools) whey, koumiss, or beef broth may be substituted. Patients in this regimen get their first soft-boiled egg after about seven days of perfectly normal temperature, and their first solid food on the 10th or 14th day. Ice cream is allowed. Clinicians who advocate this diet believe there is danger of giving too much food, and declare that cases which are even practically starved do very well. They affirm that many flare-ups in temperature, many complications and sequelæ, are due to some change in diet. Arguments against this diet are chiefly theoretical. As such a diet contains only about one-quarter of the necessary heat value, the patient must live off his own tissues, and becomes very emaciated. The return to a full diet must be very gradual, or rises in temperature, even relapses, may result. The diet described is that which practically all the patients of the Johns Hopkins Hospital have received.

Diametrically opposed to this view is the opinion of those who in addition to liquids give also solid foods—meat and vegetables—in small quantities. They declare that the mortality of their patients is no higher, that the convalescence is shorter, and that the dangers of sequelæ and relapses are less, as starvation is not added to the typhoid infection. This diet does not really differ so much as would at first glance appear from that described above, for the patient, although he may if he will, often during the fever will not take solid food. But these patients are much happier than the others during convalescence, because they do not feel starved.

Between these two views is that of those who give a soft diet (two soft-boiled eggs, a little milk toast, etc., each day) during the fever, and try to keep the heat value of the food at a point to cover the heat loss (at least 2000 to 2500 calories per day for an adult man); they give solid food as soon as it is asked for.

On one point there is more agreement—that large quantities of cold water should be given to the patient, enough to make the output of urine at least six or eight litres a day; some cases void 12 or even 20 litres. This will mean a glass of water at least every half hour, often every fifteen minutes

while the patient is awake; but he soon notes that the more he drinks the fewer baths he takes, and so drinks with heroic willingness. This "internal lavage" is most efficient in washing out the toxins of the disease.

Hydrotherapy—the cold sponge, cold pack, but always by preference the cold bath—is of the utmost value and saves six or seven of every hundred typhoid patients. The cold bath (at from 75° to 80° F.), after which the patient is left in the wet sheets for about twenty minutes, lasts twenty minutes and is given every three hours if the temperature is 102.5° F. or over; but one must not be too mechanical about this, for if the patient is very toxic, the bath is indicated at much lower temperatures. It is omitted if the patient is suspected of having perforated, or if he has hemorrhage, phlebitis, or great prostration. Often the first bath is given at 85° F., but only the first, because the reaction of these baths is not so good as that of a bath at a lower temperature. Really, 70° F. is best, but few patients stand this well. Baths are given, not to reduce the temperature (although they do make it fall after the first week), which could be more easily done by drugs, but because fewer patients thus treated die. Baths are excellent tonics to the nervous system. After them the delirium disappears, the patient feels better, his mind is keener, the lungs are in better condition, the kidneys act better, the heart is finely stimulated, the skin is better cared for. Among drugs still advised are some to reduce the temperature, many to disinfect the intestine, etc., etc., and all of doubtful value. To relieve the tympanites turpentine stupes are given, but a rectal tube introduced high and left there for some time is better. When hemorrhage has occurred or is suspected, the foot of the bed is elevated, the patient moved as little as possible, and nothing given by mouth except calcium lactate and sips of water.

Since the urine is sometimes full of the bacilli, it is customary to give all patients urotropin, five grains at least a day, and this is continued for weeks.

The nurse's problems during convalescence are especially difficult. The patients often feel starved and will soon steal solid food. Constipation should not be relieved by drugs, but by enemata, until the patient is walking about. As a rule, they sit up against a back-rest on the seventh day of

perfectly normal temperature, in a chair about four days later. They must be guarded against excitement and over-exertion. Relapses happen often enough, and the family in their efforts to explain "why" are most apt to blame the nurse.

Micrococcus Lanceolatus Pneumoniæ. ACUTE LOBAR PNEUMONIA.—Of all the acute fevers this disease deserves the most careful study, since it is one of the commonest and most fatal of them all. It is caused by one specific germ, *Micrococcus lanceolatus* (Fig. 99, *E*), an organism which has been very thoroughly studied. This germ is almost ubiquitous. It is an almost constant inhabitant of our mouths, and seems always waiting for the time when the soil of our bodies shall be favorable. At the beginning of practically every attack this germ is carried by the blood over the whole body and has its choice of practically every organ, yet it usually chooses the lung.

It will be remembered that our lungs are made up of many little lungs (Fig. 39), each an air sac communicating with the outside air through the bronchi. In the wall of the alveoli is a network of capillaries full of blood. Pneumonia is an inflammation of the walls of the alveoli. The first sign of the inflammation is that these capillaries become very congested, and blood-plasma oozes through the previously water-tight wall of the alveoli into the air space. This is the "stage of engorgement." Then into the air-cell is poured an inflammatory exudate consisting of serum, red corpuscles, and fibrine, which fill the whole air-cell with a solid red clot (Fig. 103). This is the "stage of red hepatization"—red, because of the predominance in the clot, of red blood-cells; "hepatization," because that part of the lung is now no longer a sponge of air-cells, but



FIG. 103. The adjacent margins of two lobes of the lung. The lower lobe is normal, the upper is the seat of a lobar pneumonia. Note that the air cells are filled by the exudate, the bronchi are free.

a solid organ, which feels and looks like the liver. Through the capillaries still flows the stream of blood, but now there is no air there to purify it. Next the red cells disappear, and the leucocytes migrate into the clot in large numbers, giving it a

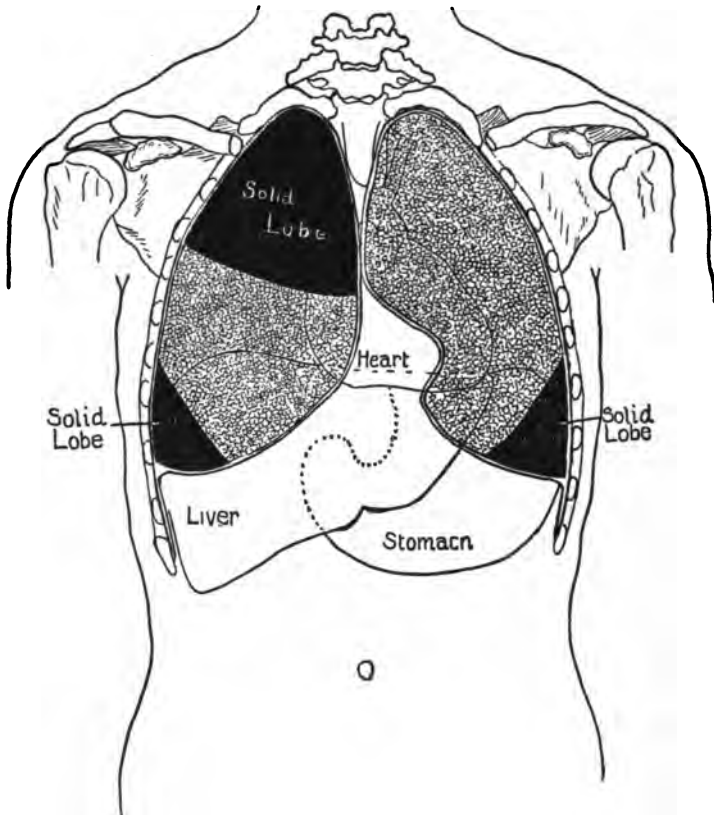


FIG. 104. Sketch of the lungs of a case of double lobar pneumonia. There is consolidation of the right upper and lower and of the left lower lobes. (Only the tips of the lower lobes can be seen in a front view.)

gray color, whence the expression, "stage of gray hepatization." Then the leucocytes in their turn die and disintegrate. But a leucocyte has within it a ferment, a substance which can digest—that is liquefy—solid substances, just as pepsin will digest meat. And just as a piece of meat is soon changed in the stomach and bowel to a fluid, so the solid clot in the

alveoli is digested—that is, is changed into a liquid. This process is called “resolution.” This liquid is absorbed into the blood (a little is expectorated) and then excreted in the urine. After the absorption is accomplished the alveolus is full of air again, in about as good condition as before the attack.

The process which we have described as taking place in one alveolus goes on in all the alveoli of one lobe, or of two or more lobes, but at least the whole of one lobe is involved, whence the name “lobar pneumonia,” given to distinguish this disease from lobular pneumonia, a condition in which many little patches of a lobe are solid from pneumonia.

The onset of an attack of pneumonia is nearly always sudden. The man who was feeling perfectly well is suddenly attacked by a terrible pain in the side which prevents his taking a long breath, and by a severe chill followed by fever. In no acute disease is a chill at onset so constant or so severe. The patient feels very ill from the first and goes to bed at once. His temperature rises rapidly and in a few days is from 104° to 106° F. He soon begins to expectorate, and the sputum is sometimes clear, sometimes very bloody. He lies in bed breathing rapidly, his cheeks flushed, his eyes bright, dilating the nostrils with each breath, and making a grunt with each expiration; the pulse is rapid and bounding; he coughs frequently—a short cough, which hurts him badly; the sputum is so sticky that he can hardly expectorate it, but it must be wiped off his lips. The sputum cup may be held upside down, but the sputum is so sticky that it will not run out. If we have to count his leucocytes we find them very numerous, from 20,000 to 100,000 per cubic millimetre. This leucocytosis is a great aid in the diagnosis. There are often fever blisters on the lips and nose.

The patient continues in this condition from three to ten days, with the straightest temperature curve seen; then comes the crisis and convalescence begins. The crisis in pneumonia is a wonderful phenomenon. Up to the time of it, the patient seems to be getting worse, seems to be even at death's door; then, within a few hours, the temperature falls to normal, the pulse and respiration slow, the cough ceases, and the patient becomes comfortable and practically out of danger. Meanwhile the lungs show very little change. They are probably

just beginning to resolve, but this sudden change for the better is not due, as far as we can see, to any change in the lungs. The fever, the rapid pulse, and all the other symptoms were due to the poison, or toxin, which the germs were forming in the lungs, and which was absorbed into the blood and carried by it throughout the body, poisoning it. The crisis doubtless means that at last the body provided enough antitoxin to get the upper hand. When this occurs the patient begins to get well. If the body cannot form enough, then the patient dies because of the general poisoning, or toxæmia, rather than because of the local condition in the lungs. The constitutional symptoms bear no relation to the amount of solid lung, for a person with part of one lung involved may be much more ill than one whose lungs are both solid, with the exception perhaps of one lobe of one lung. The question of illness depends on the person's susceptibility to the toxin of this germ.

The mental symptoms deserve especial mention. The patient is very often delirious, but there are cases in which the first sign of illness seen by friends is a mild delirium, even a maniacal insanity. Neither pneumonia nor any other fever is thought of. In this condition the patient may greatly endanger the lives of those about him, and often kills himself. Alcoholics supposed to have delirium tremens sometimes really have pneumonia.

In some cases the temperature does not fall by crisis—that is, within twelve hours, but may take from twelve to twenty-four hours to reach normal; this is called a protracted crisis. Often it takes over twenty-four hours—even three or more days; this is called a “lysis.” It may fall and then at once rise again—a “pseudocrisis.” This crisis may come as early as the third day, some say on the first day (“larval pneumonia”) but usually it comes between the seventh and tenth days and sometimes later. As a rule the consolidation resolves rapidly after the crisis, but it may take even six weeks (“delayed resolution”). If at least one lobe of each lung is solid, the case is one of “double pneumonia.” This form is more serious than a case in which even all the lobes of one lung are involved. If, as one lobe resolves, another becomes solid, the case is one of “creeping pneumonia.” When in the course of a chronic disease—Bright's disease,

for example—a rapidly fatal pneumonia develops, the case is said to have had a “terminal pneumonia.” Pneumonia often follows injuries to the chest (“traumatic pneumonia”). It is an accident following general anæsthesia for surgical operations (“ether pneumonia”), and this must be borne in mind when one is advising “perfectly harmless operations” even on young, strong persons.

As a rule, with the crisis or lysis the danger is over, for there are but few sequelæ. In some cases, however, pneumonia is followed by empyema, or by abscess of the lungs.

Although the pneumonic process usually involves a whole lobe uniformly, yet a portion may be spared. This portion is sometimes the surface of the lung, and a case of this kind is called a “central pneumonia.” Many cases are central for a day or so, before the disease reaches the pleura. There is practically always a pleurisy, since the pleura is a very thin membrane covering the inflamed lung, and this pleurisy causes the pain.

In acute lobar pneumonia we save life by guarding patients against cardiac failure, and tiding them over the toxæmia. Leave the cases alone, do practically nothing, even let them want many of the necessities of life, and over 70 per cent. will get well; do as much as we can with all the science at our command, and about 25 per cent. will die. This applies to a large hospital clinic, where poor laborers are treated. Among the well-to-do the mortality is nearer 10 per cent. But a few whose lives are in the balance we can save by measures which will help until the protective antitoxins of the body are in sufficient amount to control the situation—that is, until the patients can cure themselves. Of pneumonia patients it has been said that all the young get well, and all the old die, but there are many exceptions to this rule, as some aged patients do get well, and for very young children the disease is serious. Pneumonia is often spoken of as the “old man’s friend,” because terminal pneumonia brings his very quick and painless release, and saves him from the lingering course of a chronic disease.

Pneumonia is more prevalent in the early spring. It attacks especially the inhabitants of cities, persons of sedentary habits, those suddenly exposed to cold weather (especially half invalids), and above all others, alcoholics. It is very apt to attack a person more than once, even several times.

It is a contagious disease and may spread through a whole family, but this is rare.

The treatment is to help the natural protective agencies of the body in conquering the disease. Patients with pneumonia are the "nurse's patients," and the doctor is at hand only for general supervision. Pneumonia is a "self-limiting" disease—that is, the man gets well when his body has manufactured enough antitoxin, and by no treatment we know can we influence the course or shorten it by one day. Death is usually due to failure of the heart, which often becomes poisoned by the disease, and we can often prevent death by keeping the patient perfectly quiet and stimulating the heart. The patient should be kept as quiet as possible, without visitors, or any news which might in any way excite him. The windows of the sick room should be wide open even in winter. If possible the bed is during the day wheeled outdoors. A light flannel night-shirt should be worn. The diet may be anything suitable for an invalid, but he should drink as much water in any form as possible, for in this way much toxin is eliminated through the kidneys. Tepid, cold or ice sponges are very beneficial and may be given even every three hours. They are an excellent tonic to the nervous system, and after them the patient is clearer mentally. They should make him more comfortable. An ice-bag or warm poultice is put on the chest. Theoretically a hot flaxseed poultice is the better, and practically it is often more comfortable. Some prefer an ice-bag, but it should be one large enough to surround half the chest. Its chief object is to relieve the pleural pain. When this is very severe, either a hot-water bottle, the Paquelin cautery, or morphia is indicated. When the pulse is rapid and feeble, an ice-bag should certainly be kept over the heart. As to drugs, salts are given daily to keep the bowels open. Valuable drugs, serums, etc., do not yet, so far as we know, exist. The use of alcohol, of digitalis, and other drugs, depends on the state of the heart.

On the whole, quiet is the important thing. Absolute physical rest in bed, without any sudden movement, as rolling over or the upright posture should be enforced. Bathing, drugs, routine treatment of any kind, are not so important as this, and should be omitted rather than insisted on, if they disturb the patient physically or mentally. Often too assid-

uous ministrations are followed by sudden death from heart failure.

Genuine acute lobar pneumonia is always caused by *Micrococcus lanceolatus*, an organism which causes many other conditions also, but conditions not distinctive enough to be called "specific" diseases. Among these are septicæmia, bronchopneumonia, pleurisy, pericarditis, endocarditis, meningitis, peritonitis, synovitis, otitis, ulcers of the cornea of the eye, etc. While probably every case of lobar pneumonia is at some early stage a septicæmia, yet there are cases in which the disease is that only—that is, the organism lives and multiplies in the blood without settling in any special organ, as the lung or heart, and there setting up a local disease.

The pleurisy caused by this organism is usually purulent,—that is, an *empyema*,—and requires a surgical operation. It is the most common form, and differs, especially in children, from that due to other organisms in that the prognosis is much better.

The *endocarditis* it causes is usually of a very malignant type. *Meningitis* caused by this germ occurs especially in children and differs from the epidemic form (see page 282) in that the inflammation extends over the whole surface of the brain and is not chiefly at the base.

LOBULAR PNEUMONIA.—Lobular pneumonia differs from lobar pneumonia in that in the lobular form groups of a few alveoli, not whole lobes, are solid. A lung thus affected feels when collapsed like a soft cushion full of hard lumps, whose sizes vary from that of a pin head to that of a hen's egg. When these lumps are numerous, the lobe may be almost solid, but the consolidation is not uniform, as in acute lobar pneumonia, as these areas are of different ages, and are separated by narrow strips of normal lung. This pneumonia is usually due to the extension of an acute bronchitis to the alveoli. It is the form which in children complicates measles and whooping-cough and makes these diseases dangerous. It is an especially serious disease for babies. In adults it is a common form of terminal pneumonia in cases of blood-poisoning, influenza, meningitis, typhoid fever, endocarditis, etc.; in fact, often when we speak of a person's dying of any of these conditions it is this complication which is really the cause. In chronic consumption, the pneumonia is of this form.

Unlike true lobar pneumonia, lobular pneumonia is caused by a variety of organisms; by which one we cannot discover from the symptoms. Nor do we know, when the patient has a fever due to a certain germ—typhoid fever, for instance—whether or not this complication is due to that same germ. It sometimes is, but sometimes it is due to a secondary infection; that is, the first infection has so lowered the resistance of the patient that other and commoner germs can gain a foothold. The treatment is that of acute lobar pneumonia.

Bacillus Tuberculosis. ACUTE TUBERCULOUS PNEUMONIA.—This form of tuberculosis at first resembles closely acute lobar pneumonia. But in acute lobar pneumonia the cause is *Micrococcus lanceolatus*; one or more lobes are solidified, as all their air-cells are filled with a clot of exudate consisting of fibrine, red blood-corpuscles, and leucocytes; this exudate is the result of inflammation of the walls of the alveoli, but these walls, although inflamed, are not seriously affected, and the blood flows as before through the network of capillaries in them; later the exudate is liquefied and absorbed, all signs of inflammation disappear, and the air-cells are as normal as before. In acute tuberculous pneumonia the air-cells of one lobe or more are filled with an exudate similar to that of acute lobar pneumonia, but caused by another germ, *Bacillus tuberculosis*, and the toxin of this is far more poisonous than is that of *Micrococcus lanceolatus*, for it kills the walls of the alveoli, and the cells of the exudate, so that soon the solid lobe is a solid mass of dead matter. Cut through it, and the cross-section, because of its combination of white, gray, and green colors, resembles some beautiful green marbles. Soon this dead mass begins to liquefy and not only the exudate, but the living tissue also, and is removed through the bronchi as sputum, leaving behind a hole, or "cavity" in the lung, the size of which depends on the amount of lung which died and the duration of the disease; for some die before any softening begins, and some live till there is practically no lung left—only a hollow, empty, pleural sac. Some get fairly well, but the cavity remains, or, if it is obliterated by contraction of its walls, its scar is always there.

The diagnosis at first is acute lobar pneumonia. This is an almost unavoidable mistake, unless the patient is known

to have had a chronic cough or other symptoms of chronic consumption before this attack began, for tuberculous pneumonia is often an acute outbreak of a chronic tuberculosis. With this diagnosis and a hopeful prognosis, all goes well till the eighth, tenth or even the fourteenth day, and still there is no sign of a crisis or lysis. Then the sputum becomes yellower, and the temperature more irregular; the patient begins to fail, and soon the tubercle bacillus is found in the sputum.

A Tubercle.—A tubercle, a little tumor, a little hard lump, is the characteristic lesion of tuberculosis, and that from which the disease gets its name. When one or a few tubercle bacilli lodge in an air-cell or bronchus, for instance, they may be killed at once by the tissue fluids, but too often they successfully resist these, and so the cells in the neighborhood try to “choke them” to death. That is, the lung quickly builds a little tumor, just big enough to be seen with the naked eye, around the bacilli. Meanwhile the few have multiplied and now there are many there. The bacilli are thus, as it were, “rolled up in a mass of tissue,” or “surrounded by a wall.”

The result may be that the bacilli are in this way killed, and the tubercle remains as a little tumor, like a grain of sand in the soft lung. But, as a rule, the poison of the germ wins, and the whole tubercle caseates—that is, dies and becomes a little lump of cheesy or clay-like matter, conspicuous in the normal lung as a yellow dot. But the lung usually perseveres and builds a new wall of tissue around the caseous mass. This may stop the process; then the cheesy matter will become calcified—that is, impregnated with lime salts and turned into limestone. As a rule, however, the bacilli

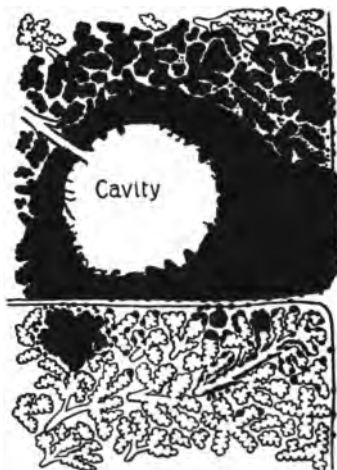


FIG. 105. This figure represents the adjacent edges of two lobes of a lung. There are miliary tubercles in the lower lobe, and conglomerate tubercles in the upper. In the upper there is exudate, and destruction of air cells and bronchi alike.

spread, new tubercles form in the wall of the dead tubercles, and so on, till large masses, as large as one's fist, of tubercle and scar tissue are formed. These are called "conglomerate" tubercles. There is no normal lung tissue in these masses;

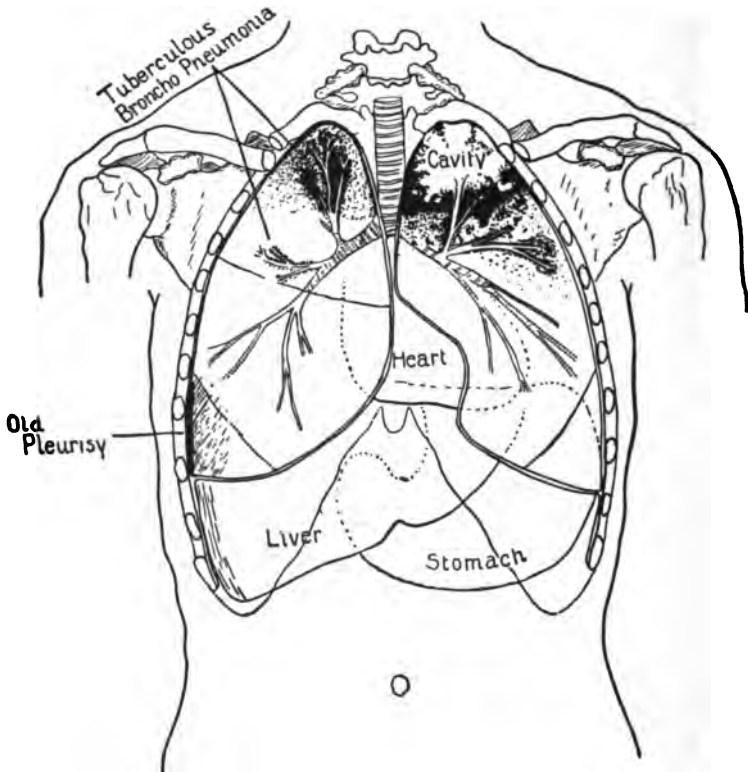


FIG. 106. A Diagram of the chest, showing tuberculosis of both lungs. The oldest tuberculous trouble is the pleurisy of the right lower axilla. This has healed, but has left a patch of "thickened pleura" and slight fibrous changes in the lung. The most recent lesion is the slight bronchopneumonia at the base of the right and left upper lobes. There is a much more extensive tuberculous pneumonia at the apex of the right upper lobe. At the apex of the left upper lobe is very extensive tuberculous pneumonia, with cavity formation. Scattered areas of beginning bronchopneumonia and scattered miliary tubercles are represented in the lower part of this lobe.

they consist entirely of tubercles, scar tissue, and caseous matter. The tissue in the centre of these large masses, lung tissue and all, usually dies, and the dead matter is expectorated through the bronchi, leaving a cavity in the lung. All this time the lung keeps on building a wall around these masses

of tubercles. The tuberculous material may all be expectorated, and then the cavity with its wall will remain. But the scar tissue always contracts, and the cavity may become much smaller. But quite as often new tubercles start up in this protective wall, and so the disease spreads on.

Tuberculosis always leaves scars behind. In the lung years later can be found these puckered scars, or cavities, almost closed by the contraction of their thick wall, or calcified masses. If these scars are overlooked, the lymph-glands along the bronchi will tell the story, for they will be large caseous or calcified masses. One remembers that the lymph-glands along the lymph-vessels are filters for the tissue lymph, and it is in this that the bacilli live; hence these glands must bear the brunt of an infection.

CHRONIC PULMONARY TUBERCULOSIS.—This slow formation of conglomerate tubercles and the slow cavity formation are characteristic of the common form of consumption. It is very insidious in its onset and course. The patient usually has been treated for a dozen diseases before the correct diagnosis is made, which is very unfortunate, as it is much easier to cure the disease early in the course than it is to cure it later on.

Symptoms at Onset.—Suppose that a few of the tubercle bacilli lodge at the apex of one lung,—their favorite location,—in the walls of the smaller bronchi. A few tubercles form around them; a few alveoli are filled with exudate; the process slowly spreads from this as centre. What will the symptoms be? In the first place the patient may not notice a single symptom until the consumption has reached a fairly advanced stage. Cases with “latent onset” are not so very rare. But the chances are that he will notice that he is losing weight, that he is not quite so strong as before, that he is a little paler than usual, that he has a little indigestion and a poor appetite. He certainly will not realize, until someone tells him of it, that he coughs or at least “clears his throat” every morning just after rising. He may or may not expectorate what is raised, but if he does, it is “only slime,” “only mucus from the throat.” He will perhaps feel rather tired afternoons between four and six o’clock, but in the morning he feels in fine condition. If by any accident his temperature is taken, the chances are that it is found to be subnormal

(96.5° to 98.4° F.) in the morning and from 99° to 99.5° in the afternoon. "No fever, merely a high normal temperature," he will say. He surely will take some medicine now. Perhaps his paleness is marked, so that a diagnosis of anæmia is made. Then iron in good doses, and some "tonic" are given to him. Perhaps his loss of appetite and his indigestion are what trouble him most, and so his stomach is treated. His indigestion may be extreme; he may vomit every meal; he may have severe pain after eating; he may loathe the sight of food. It is very difficult to persuade this man that his stomach is perfectly normal; and yet such is often the case. Or perhaps his trouble began with a cold, which "has hung on." He knows that he does cough a little every morning, but it is only a case of "neglected cold," of "chronic bronchitis," a "winter cough"—nothing more. The slight amount of innocent-looking sputum does not worry him. Or perhaps he has let the trouble run along a little longer than we have been supposing. He has chilly feelings in the afternoon, or a slight sweat at night. The chances are that he will be sure it is malaria and will begin taking quinine. Or perhaps he has a slight touch of pleurisy, a stitch in the side, or a little water on the chest. He consoles himself with the reflection that it is only "pleurisy." Perhaps he has noticed that he is getting hoarse, possibly it also hurts him to swallow; "chronic laryngitis" is his consoling thought. Perhaps the glands in his neck get large and stay so; yet this does not worry him; "scrofula," he says. Or possibly while "in the best of health" he has a sudden desire to cough, and expectorates a mouthful of bright red blood. This last man is fortunate, for, although his case need not be so advanced as many cases of the kind described above, he will be so thoroughly frightened that he will be ready to obey orders, and so he will have a better chance to get well than such a man as described above. Now probably there are to-day in every large city hundreds of patients who are treating themselves, or are being treated, for one of the above mentioned diseases, and yet their real trouble is a little spot in some part of the lungs, where it gives them no local symptoms. There the disease is either spreading slowly, or is being slowly defeated by the self-protecting agencies of the body. In over one-half the cases, probably the correct diagnosis will never

be made. The patients will get well, thanks to their own inherited "*vis medicatrix naturæ*," no thanks to any of the expensive tonics they have been taking, but at autopsy, years later, we find the tell-tale scars.

Usually, after a few months of treatment, during which the diagnosis and hence the medicines have been changed several times, tuberculosis cases become convinced of the true nature of their trouble. The cough has become frequent and troublesome; there is no doubt as to the afternoon fever; the patient has night-sweats; he loses weight and strength more rapidly, and he is obliged to admit that he is "going into a decline" or that he "has a chronic cough, which may turn into consumption if he doesn't check it at once." So more cough medicine is taken. Finally he admits that now at least he has consumption. "*Has consumption?*" He has had it a long time, even before that day when months ago he first noticed a little paleness, dyspepsia, or loss of weight; but such is the optimism of tuberculosis patients, such the effect of the euphemisms used by their friends, that the disease has had months in which to get good headway—now it claims its victim.

Hemorrhages are frequent accidents of chronic tuberculosis. They sometimes occur very early in the disease, and they may even be the first symptoms. Small in amount, they are never serious, and they usually serve the purpose of frightening the patient into taking good care of himself. Some cases of the "hemorrhagic type" have hemorrhage after hemorrhage during the whole course. The most important hemorrhages occur when the patient has almost recovered from consumption. They are usually profuse, often fatal, and they come very unexpectedly. They are not due to the activity of the tuberculosis, but to the injury inflicted on the arteries during cavity formation. Arteries have very resistant walls, and some of them instead of becoming thrombosed when the lung is decaying round them, remain channels for the circulation and lie exposed in the wall of the cavity, or cross it, exposed on all sides. But at some points the wall itself becomes weakened, tiny aneurisms form, and the future history of such cases depends on these aneurisms rather than on the consumption.

When a person suddenly begins to bleed from the mouth,

the question of the source of the blood is important. The blood may come from the lungs, as in consumption, or from the stomach, as in a case of gastric ulcer. In the latter case it is vomited rather than coughed up, although with vomiting there is often considerable coughing, and vice versa. The blood from the stomach is usually dark in color and often mixed with food, while in lung cases it is bright red and frothy. In mitral heart disease, in cases with pulmonary infarction, and from ulcers of the larynx there may be rather profuse hemorrhages. In aneurism of the aorta, a rapidly fatal hemorrhage occurs if the aneurism ruptures into a bronchus. There are several other causes of hemorrhage, but these are the most common.

The *course* of a case of chronic pulmonary tuberculosis is variable. The tuberculous bronchopneumonia may progress rapidly, cavities rapidly form, the fever run high and irregularly, with daily chills, until in a few weeks the patient dies of "galloping consumption." Or the patient with less marked symptoms may feel now better, now worse; now he may be apparently well, now ill in bed. So it goes for months and years, until an acute exacerbation of the trouble, often a tuberculous pneumonia, ends the scene. Sometimes the patient "gets used to his disease." He feels well. If a laborer he is able to do a hard day's work and keep it up for years. Yet he has a chronic cough with slight expectoration, and is always spreading the disease among those more susceptible than himself. He may live fifteen or twenty or more years, and he will probably have an opportunity to attend the funerals of some of the doctors who have told him of his consumption, and have warned him to begin treatment at once. But he will attend the funerals of relatives also, especially of members of his own family, and he will little think that he is directly responsible for these deaths; that with that small amount of sputum, which didn't bother him at all, and which he carelessly expectorated, he has killed them just as truly as if he had wilfully put poison in their food. But such is often the case and such often are the results.

The *diagnosis* of these cases rests chiefly with the sputum. Even at the very first the patient can, if he will, save the material which he raises when he clears his throat in the morning, and in it can usually be found, even so early (it may

take several trials) the germ which when seen should not be mistaken. Even very early traces of the disease can be found by examining the lungs. The temperature chart will help, and even at the very first, as soon as the diagnosis is made, active treatment should begin.

The tubercle bacillus, or *Bacillus tuberculosis* (Fig. 100), is one of the germs concerning which we know most. The diagnosis of tuberculosis is not now made unless this germ is found, or unmistakable evidence of its presence is obtained. A reasonably well-trained man should not mistake it. One judges it by its size and shape, but especially by the "fast colors" it assumes when stained with certain dyes and then treated with strong acids that will bleach almost every other germ.

The tubercle bacilli are fairly easy to find in cases of consumption. The sputum is often full of them; an average case of consumption will expectorate from one to four billions of these germs every day. Sputum containing them is very dangerous while wet, if we get it on our hands, and after it becomes dry and is blown about as dust, there may then be some danger. Also the particles of moisture which a consumptive expels when he coughs must contain many of these germs. These bacilli are scattered in every place where men are expectorating carelessly. The probability is that we are inhaling them very frequently. Why, then, are we not all consumptives? is of course the question that at once arises. In the first place, because nearly all of these germs are dead when we inhale them, killed by that cheapest and best disinfectant, sunlight. Wherever the sun's rays can shine, in that place there is little danger from these germs. In the second place, the soil (our bodies) into which these germs, seeds of disease, fall, must be a suitable one, else they are quickly killed by the protective agents in our tissues. We almost never inherit this disease, but we can inherit a soil which is favorable to its germ, and if the air in a house is always full of dust laden with these germs what chance has the baby? This soil is determined especially by the mother, for the children of a consumptive mother have by no means the chance to combat this disease which those of healthy mothers have. The soil is made favorable or hostile by our manner of life also. These germs find a poor soil in the man who lives

an active, outdoor life with plenty of exercise, food and sleep. The soil seems especially favorable for the germ after an attack of measles, whooping-cough, typhoid fever, smallpox, diabetes, and during any chronic disease, such as heart, kidney or liver trouble. In fact, the case may be truly stated as follows: we are all repeatedly exposed; the great majority of mankind have somewhere in the body before they die at least a small focus of this disease; about one-third of all persons have at some time during their lives symptoms from this focus—perhaps a lung trouble or a pleurisy or enlarged glands or some bone or joint trouble; while one in every ten human beings dies of it. Of course every one must die at some time or other, but this disease seizes especially those between the ages of 18 and 35—that is, those in the prime of life.

Tuberculosis in its various forms kills more persons than any other one disease—about 150,000 a year in our country alone. And yet the saddest, as well as most hopeful, aspect of the whole thing is that were only ordinary rules of cleanliness observed our grandchildren would not know of the disease, except as a matter of history. To cure some disease we may properly search for serums, antitoxins, and wonderful medicines, but to stamp out this disease, the worst of all, will take only cleanliness. The disease is caused by germs spread by a patient. Whether they reach the lung through the bronchial tree, or, whether the tonsils are first diseased, or whether we swallow them in milk and other foods is of little consequence; we get them from a person with tuberculosis, and he need not have spread them. The germ gets into the body. It need not cause any trouble at the point where it enters, but it is carried around in the blood or lymph stream and finally finds a spot where it can grow—a bone, a joint, lymph-glands in children, the lungs, especially in adults; any organ in our whole anatomy may be infected. Here it settles and here it multiplies, and here may be formed a focus of disease. But the germ may remain quiescent for years in the spot where it settles, and then suddenly become active and cause disease.

These bacilli are short-lived in the open air; in dark, damp rooms they may live for weeks. It is probably no exaggeration to say that if for two generations consumptives

should cough into handkerchiefs and expectorate only into proper receptacles, and these should be properly cared for, the third generation would know the disease only by name. Probably the disease would rapidly disappear from cattle also, and thus this source of danger would be removed.

Individual Prophylaxis.—The person with consumption need not be dangerous, but often is. A few cases in point are mentioned in Osler's text-book. In one physician's practice one chronic consumptive had buried four wives, one three, and four two. Some day the laws will recognize such deaths as cases of homicide. The wives would not have died had their husbands been decently cleanly. The consumptive should train himself to hold always a handkerchief before his face as he coughs. He should always expectorate into a suitable sputum cup—one which can be burned with all its contents, boiled or disinfected with strong fluids. He should remember that his sputum is poisonous and should treat it as such. The urine and stools may contain many bacilli, but these are usually better cared for. Dark rooms should be avoided. It is no wonder that there is most tuberculosis among those who live in alleys and among the inmates of convents and prisons; in such places the death-rate from this disease may be 75 per cent. instead of 10 per cent. A family moving into a new house should always know whether or not a consumptive has lived there before them, and if one has, the whole house should be properly fumigated, cleaned, repapered, and repainted. One case will illustrate this. A man in the employ of this hospital (the Johns Hopkins) died of chronic consumption. The health officials fumigated the room in which he died, oblivious of the fact that he had lived for months "in the whole house." Then another employé of this hospital moved into that house with his wife and four children, all in good health. In six months the wife and three children had died of consumption, later the baby died, and now the husband has a chronic cough. This is not an unusual case. Tuberculosis is above all else a house disease, and houses which have one case now will usually be found to have been the home of several cases in the past, or will become such in the future. The sputum on the walls or floor of a poorly lighted room can retain its virulence for six weeks or more. Who next will move into that house?

Persons who fear consumption should plan to work and live as much in the open air as possible, should sleep and eat as much as they can, should sleep with the windows wide open, and should take a cold bath or cold plunge every morning. The prognosis will depend first on the soil inherited. Some with advanced disease can get fairly well, especially those who have inherited a soil hostile to the tuberculosis germ—that is, have no tuberculosis in their family history. When patients have a bad family history, the prognosis is doubtful, no matter how slight the lesion now, or how vigorous the fight they make for life; yet fight they should, for many win. Next, the outlook will depend on the stage of the disease when it is discovered, and those with an early diagnosis have a much better chance than those who temporize under the hope of a different diagnosis. Finally, the outlook will depend on the vigor and conscientiousness with which the treatment is followed. No matter how early the case, the patient should give up all work and for at least six months should make the struggle for recovery his sole occupation.

The treatment is more a social question than a medical one. The doctor can make the early diagnosis, but after that the cure rests with the patient, the nurse, and the friends. This treatment consists of rest and of freedom from labor and if possible, from worry. Just so long as there is fever the patient should stay in bed. When there is no fever, and he is up, he should govern all his acts with reference to the treatment. There is no heroism in staying at work until one has to give up, but a foolish bravery, which too often results in death.

Fresh air and sunlight are the great curative measures. The air should be dry air if possible, at a high altitude if convenient, but at least it should be fresh. While the patient is staying in bed, bed and all should be wheeled outdoors during the day, and during the night all the windows should be wide open, no matter how cold it is or how much rain or snow blows in. Clothed in proper flannels, a warm cap, and mittens, the patient soon enjoys such a life. When up, the patient must sit out or walk out in the open air, no matter what the weather. The question often arises, "Is it necessary to go to a sanitarium?" And the answer is, "No, not necessarily," although, if the patient can afford it, it is much better. The class work

among the poor consumptives in our crowded cities, especially Boston, has shown about as good results as have the most expensive sanatoria. There is some advantage in mountain air, but the chief advantage of a sanitarium is the discipline which the patient is made to feel and obey. It is seldom that consumptives are willing at first to do what is best for them, since, unfortunately, these things are not the most comfortable, and the rigorous routine of a well-organized institution does help in forming right habits, and the patient should stay until he has begun to enjoy those features of the treatment which were at first so disagreeable to him. In the city the small tent on the roof or in the back yard, in which the poor patient sleeps, is the salvation of many cases, and the patient is made to keep a record of the number of hours spent outdoors each day. The next point of importance is the food. It has been found that the more a patient eats, the better are his chances of stemming the tide of the disease. But eating is often a hardship, for in the majority of cases loss of appetite and dyspepsia are two frequent features of the disease, even in its earliest stages. Here again the authority of the institution counts, especially one so heartless that such a dialogue as the following can be heard within its walls.

"Here is a good large meal for you."

"But I don't want it."

"Never mind. You must eat it."

"But I certainly shall vomit it all up."

"Never mind. We shall prepare you another."

"But I *will not* eat it."

"All right. Then we shall use a stomach pump and pour the food into your stomach."

Such treatment seems heartless, but it is necessary. The patient's appetite is "delicate." He demands the "dainty" things, and family and friends sacrifice much to provide them. That is one sad feature of these cases. If not warned and advised, the relatives will keep the patient in the nice, warm room which he enjoys so much, will shield him from every draught, will feed him with the most expensive things their means allow. We have but one remark to make about this condition. If the family were to deliberately plot against the consumptive's life, and plan to put him out of existence as soon as possible, they could not, without resorting to poison

or violence, choose a better way than that which we have described. Again, the patient demands to stay at home. He rebels at the hospital. He wishes to die with his family, and he does so; or, more correctly, the whole family dies with or soon after him. Nature seems to have omitted only one precaution in this plan for the extermination of the unfit—she has not provided any way of burning down the house after the last member of the family dies. Of the foods most important, milk, meat, and eggs stand at the head of the list.

Of medicines there is little need. Perhaps the physician will give cod-liver oil (especially to children to keep them well nourished), some simple tonic to improve the appetite, cold baths when the fever is high, some simple remedy to make the cough less distressing or to lessen the night-sweats, some creosote perhaps, but to combat the disease there is as yet no drug, no serum, no "cure." The market is full of such remedies and the advertisements promise astonishing results, but the actual result is the impoverishment of the patient, so far as his money goes, and the loss of valuable time which should be spent in combating the disease.

It is very good news that since this rational prophylaxis and treatment were begun tuberculosis has diminished. The death-rate from it in some cities is only one-half what it was; thanks to the crusade against tuberculosis, the average age of Americans has been increased by over seven years; while of the cases 80 per cent. who take the treatment thoroughly, now become "arrested" cases and able to return to active life. The former opinion no longer prevails that the patient "goes the way of all consumptives—to an early grave."

When a *hemorrhage* occurs there is usually great excitement, and often many foolish things are done. If it is a small hemorrhage, the patient should be quickly got into bed and kept as quiet as possible. He should lie on the diseased side if we know which it is. The foot of the bed should be elevated. For a day food is withheld, but cracked ice is allowed. Later light, stimulating food is given. Of drugs, opium, aconite, calcium lactate, and purges are the best; opium keeps the patient quiet and relieves the cough. Aconite and purgation reduce the blood pressure in the lungs and calcium lactate increases the coagulability of the blood. The profuse hemorrhages of ruptured aneurisms or opened arteries are often

rapidly fatal, and all one can do in such a case is to keep the patient perfectly quiet for a few hours. If he faints, let him alone. This lowers the blood pressure and is very beneficial. Opium is not given in cases of profuse hemorrhage, as we wish the patient to cough the blood up.

Although the *cough* may be troublesome, it is necessary in order to remove the sputum. But since a tuberculous ulcer of the larynx is often the cause of a very irritating cough, the throat should be examined and, if necessary, treated. An irritating bronchial cough can be relieved by inhalations (equal parts of creosote, turpentine, and benzoin; one tablespoonful in a pint of boiling water). A warm poultice on the chest will often give relief. In the morning a glass of hot water containing fifteen grains of bicarbonate of soda will often aid. Medicines to relieve the cough—cough syrup, etc.—usually contain opium and should be used only under the doctor's directions. The night-sweats are less distressing when the patient uses flannel night clothes.

TUBERCULOSIS OF THE LYMPH-GLANDS.—The lymph-glands are minute filters shaped somewhat like a kernel of wheat and scattered along lymph vessels. It will be remembered that the lymph-vessels drain the fluid in which the body-cells lie, and hence contain much of their ashes. They also drain away the products of disease—toxins and germs. The lymph-glands are the filters that remove these and thus protect the body. That is the reason why, when we have an infected finger, we are often sore in the armpit; the glands in that place are fighting the germs. But often the glands themselves are overcome in this struggle, and an abscess forms in them.

In tuberculosis the glands are themselves the seat of the disease. They swell often to the size of lima beans, but, since they are in groups, the masses of glands may be fist size. Sometimes the glands win,—kill off the germs in them,—and slowly return to normal size. More often the centre of the gland becomes caseous and remains throughout life a calcified mass. Often the glands suppurate—that is, become an abscess, and the pus must find some outlet. Often the local disease of these glands is in itself not very important, but so frequently is the infected gland the starting-point of an acute miliary tuberculosis that it becomes a very important

matter. Sometimes all the lymph-glands of the body are affected, but this condition is rare. The glands most often involved are those of the neck, and of the mesentery of the intestine.

Tuberculosis of the glands of the neck is the most common form of this disease, especially in children. The popular names of this condition are "scrofula" and "kernels in the neck." These glands often become diseased, because they drain the mouth and tonsils. The tubercle bacillus is often in our mouth, but is quickly destroyed by the protective substances of the body. The troubles which lower the resisting powers of children and allow these bacilli to get foothold are nasal catarrh, chronic throat catarrh, enlarged tonsils and adenoids. It is probable that the tonsils are often tuberculous. Since there is good reason for believing that many a case of pulmonary consumption at the apex of the lung is only a step further in this chain of infection—the tonsils, the glands of the neck, the lung—the proper care of a child's nose, throat, and tonsils is strongly urged. For the most part the kernels disappear, though some may be left as hard, calcified lumps; but very often suppuration begins. The bunch in the neck was composed of separate glands, but now, when suppuration occurs, they mat together and become soft; an abscess "gathers" and breaks, leaving a sinus, or "running sore," which may remain open for months and then close, leaving the ugly scars so often seen in the neck.

The bronchial lymph-glands drain the lung, and so are soon infected in pulmonary tuberculosis. They cause no symptoms unless they get so large that they press on the trachea, blood-vessels, or nerves in the chest, or unless an abscess forms.

The glands of the mesentery and those behind the peritoneal cavity, hence on the spine and at its sides, drain the intestines, and since we often swallow tubercle bacilli in milk and other food, are frequently tuberculous. This condition occurs especially in children with intestinal catarrh. Sometimes these glands are the only organs affected and form large masses, a condition sometimes called "consumption of the bowels" (although the intestines are not diseased), or "tabes mesenterica." Important symptoms are a gradual loss of strength and a wasting away of the patient. They are

due chiefly to the disease, but partly to starvation. The fat, which is absorbed by the lymph-vessels now blocked by the diseased glands, is lost in the stools. The abdomen is much distended and there is diarrhœa with thin, offensive, fatty stools.

The treatment of tuberculosis of the lymph-glands of the neck is the same as that of pulmonary consumption, and it is remarkable how rapidly large glands will disappear. If they have suppurated, of course the abscess must be opened. A few years ago the tendency was to excise these glands, but now this is no longer the practice.

ACUTE MILIARY TUBERCULOSIS.—This tuberculosis, which is really tuberculous septicæmia, is the worst form of the disease. Its beginning is supposed to be the rupture of a tuberculous focus directly into a vein. This tuberculous focus may be a blood-vessel wall, or a caseous lymph-gland, in a person who never suspected he had any disease, or some other focus which sloughs directly into a vein; sometimes there is a tuberculous disease of the wall of the thoracic duct itself. In any case the bacilli are poured from this focus directly into the blood stream, and are carried all over the body, starting up myriads of tiny tubercles, sometimes all over the body, sometimes in one or a few organs, but practically none large enough to be detected before the autopsy; hence the name "miliary" tuberculosis. In children these may form many large tuberculous masses.

It is one of the hardest fevers to diagnose, unless it is known that the patient has had a chronic tuberculosis. Many a case is an almost exact clinical picture of typhoid fever, without, of course, the specific symptoms. Some cases start as cerebrospinal meningitis; some, as very severe bronchitis. The patient is very ill from the first, with high, irregular fever, and after a few days or weeks of illness dies. There is no treatment except to make the patient comfortable.

Somewhat akin to the form of tuberculosis described above is the *tuberculous toxæmia*, which results from the absorption of large quantities of tuberculous poison from some focus, often hidden, without (to any degree at least) a pouring out of the organisms themselves from the focus into the blood. These patients recover. Some of these cases cannot be diagnosed and appear on our records among the cases entered

as "continued fever." It is likely that before typhoid fever could be absolutely diagnosticated, they were nearly all grouped under that heading, and that this explains the experience of those who declare that they have had typhoid fever more than once; for "clinically" (without laboratory aid) the two diseases cannot be distinguished. This disease often recurs several times, but, as a rule, in the later attacks the tuberculous nature is evident from the appearance of some focus, usually in the lungs.

TUBERCULOUS MENINGITIS.—This form of tuberculosis is popularly known as "water on the brain." It is common in children, although it also attacks adults, in which cases it is usually more acute. It is a form of acute miliary tuberculosis with especial localization of the tubercles in the meninges (membranes of the brain).

The child has usually been rather ill for weeks; perhaps he is known to have a chronic tuberculosis; sometimes the meningitis follows measles or whooping-cough. He loses weight and strength, becomes very irritable, and may show a complete change of disposition. Then suddenly begins an acute illness which in the initial stage, or "stage of irritation", is marked by fever, vomiting, and headache, and sometimes by convulsions. The cries of the child, sometimes occasional, sometimes continuous, are very piercing and indicate a terrible headache. The pulse is now slow and irregular. Gradually the cries cease. The child becomes dull, and hard to rouse. His emaciation is conspicuous. Finally the "stage of paralysis" begins, with paralysis of a few muscles (that of the eye is important) or of most of the body. The neck and back are often stiff, the head retracted. Convulsions, sometimes slight, sometimes severe, are not uncommon. The mental dulness deepens into coma, and soon the patient dies.

The diagnosis is made by lumbar puncture, as the tubercle bacillus is quite easily found in the spinal fluid; but even without this test one is seldom in doubt as to the condition.

The outlook is practically hopeless. The treatment is to make the patient as comfortable as possible while conscious, and to keep up the nutrition by sufficient feeding. The lumbar puncture is sometimes made daily, as so many of the symptoms are due to the high pressure around the brain, which is relieved by removing some of the fluid.

TUBERCULOSIS OF THE SEROUS MEMBRANES.—The serous membranes are thin, have very smooth, glistening surfaces, and line the cavities of the body, and cover the organs which lie in them. These cavities are such only in name, for normally there are no cavities at all, as the organs which they contain completely fill them. The serous membranes, always moist with serum, prevent the friction which would naturally result from the constant rubbing of these organs against each other and against the walls of these cavities. The cavities to which we now refer are the pleural, pericardial, and peritoneal.

The pleural cavities, two in number and not at all connected, contain the lungs. The lungs lie free except at the point where the bronchi and blood-vessels enter. The rest of the lung surface is covered by a serous membrane, the visceral pleura, and this pleura everywhere rubs against the parietal pleura, which covers the inner surface of the wall. These two opposing surfaces should rub with perfect freedom, for the lung expands considerably during inspiration and collapses during expiration. The lung is always inflated and "on the stretch," as there is no communication between the outer air and the pleural cavity and this cavity contains no air; hence the atmospheric pressure within the lungs will always keep them expanded. But make a hole through the chest wall down to the lung, opening the outer pleura so that air can get between these two pleuræ, and the lung will collapse like a punctured balloon down to almost one-sixth of its usual size. Then there is pleural "cavity" enough, and the condition is called pneumothorax. This may be caused by a stab or a bullet wound, either of which opens the pleural cavity from without; but in the vast majority of cases it is due to a tubercle under the pleura, which by ulceration causes a communication between a bronchus and the pleural cavity.

The pericardium is the closed sac containing the heart. With each beat the heart contracts and expands. It is attached at only one point and lies free in a sac whose inner surface, like the heart's outer surface, is covered with smooth, serous membrane, which can rub without friction. Here also the word "cavity" is misleading, for there is no empty space between these two serous surfaces until we open the outer surface and let air or fluid enter.

The abdominal cavity is everywhere lined with the peritoneum, a serous membrane, and all the organs within it—liver, stomach and bowels, and spleen—are covered with this membrane. These organs are in almost constant motion and should move freely without friction. On the other hand, the kidneys, pancreas, aorta, etc., lie behind and not in this cavity. Here also there is no true cavity until you open the outer wall. It is true that the stomach and bowels are hollow organs with much empty space often filled with air, but the air in the stomach is not in the peritoneal cavity.

These serous surfaces are everywhere identical in structure and are affected by the same diseases. Yet so little communication have they that in only two diseases—tuberculosis and cancer—does infection of one travel easily to the others.

TUBERCULOUS POLYSEROSITIS.—Tuberculous polyserositis means tuberculous disease of any one or all of these serous sacs. It may be acute, subacute, or chronic. In acute inflammation tiny tubercles spring up over the serous membrane, spoiling its smooth, glossy surface; flakes of fibrine and pus cell exude, and stick the two pleural surfaces which are in contact tightly together; then these fibrine “adhesions” become permanent—that is, are replaced by scar tissue—so that now the two surfaces which should rub together without friction are inseparably joined together—are “adherent.” Thus the acute disease gets well, but leaves a chronic trouble—these adhesions—behind. There is now truly no “cavity” left; it is entirely “obliterated.” But sometimes, and this is the rule in tuberculous serositis, the result of the inflammation is a large amount of fluid, clear yellow blood-serum usually, although when the trouble is very acute, it is red because of red corpuscles; when there are many pus-cells it is opaque yellow. When the opposing pleural surfaces are not adherent this fluid can distribute itself among the organs as gravity dictates. It makes room for itself in the abdomen by compressing some organs, as the stomach and bowels and distending the abdominal walls, and in the chest by allowing the lungs to collapse. If a patient recovers without treatment from this form of tuberculosis, the fluid is reabsorbed. Sometimes the serous surfaces become smooth again, but more often some adhesions are left.

In another and more chronic form only a little fluid col-

lects, but the serous membranes become thickened, and in them form large conglomerate tubercles, which may become cheesy. In the most chronic form there is no exudate at all, but the membranes become thicker and thicker because of a great growth of connective tissue in them, and finally, instead of being about as thick as paper, they are even a quarter of an inch thick. This thick membrane is widespread. It is due to a very chronic form of tuberculosis. This thick layer of scar tissue contracts, of course, and may greatly deform the organ encased by it.

The symptoms of tuberculous polyserositis will depend on the toxæmia of the general disease and on the local disturbances of the organs in the cavities involved.

Acute tuberculous pleurisy may begin usually with a chill, fever, a dry cough, and a "stitch in the side." Every motion of the lungs that makes the inflamed pleuræ rub over each other is attended by an exquisite pain, which cuts short every respiration. But the majority of cases, especially those with effusion, are latent, and the date of their onset cannot be stated. The patient may continue at work, although he feels badly and is losing weight and strength. When the pleural cavities, one or both, contain much fluid, the patient will be short of breath, as the collapsed lung does not "ventilate well;" but it is remarkable how much fluid can accumulate, when this process is slow, without the patient's having any symptoms suggesting the chest. He is often treated for stomach trouble, cancer, or heart disease, until death. This is a most calamitous mistake since pleurisy with effusion, if treated properly, is practically never fatal. The majority of cases have no further trouble after the acute pleurisy subsides, but those with many adhesions often have symptoms of greater or less severity. As the scar tissue of the adhesions slowly contracts, the chest wall is drawn in, the shoulder is drawn down, and the spine usually is curved laterally. This condition is called "chronic dry pleurisy," or "contracted chest." There are usually pains—sometimes very severe pains in the chest, and the patient is rather short of breath. Since his lung is more confined and less expanded than normal during inspiration, it is more susceptible to other diseases. During convalescence from the acute trouble this patient should take regular breathing exercises to prevent

in some measure the contraction. One can get the best exercise by sitting in a chair, grasping with the hand of the well side the round just below the seat, and pulling hard on this during each inspiration. This allows only the affected lung to expand. One objection to most such exercises is

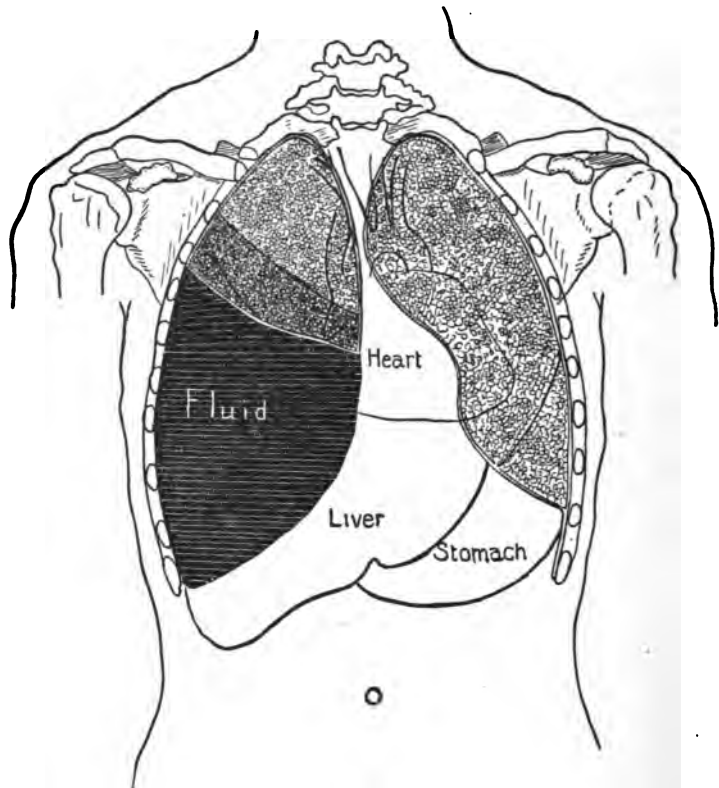


FIG. 107. Diagram of the chest of a case with pleurisy with effusion on the right side. Note that the lung is above the fluid, and that the liver is lower than normal.

that the well lung does most of the work. When we say that patients recovering from acute pleurisy with effusion seldom have further trouble, we mean pleural trouble. They do later often develop consumption, and this is one of the proofs that the pleurisy was tuberculous. Examination of the fluid itself will often show this conclusively.

The sharp pains of acute pleurisy are relieved by strapping the chest with a tight binder or a broad strip of adhesive plaster, by local applications of heat—the Paquelin cautery,

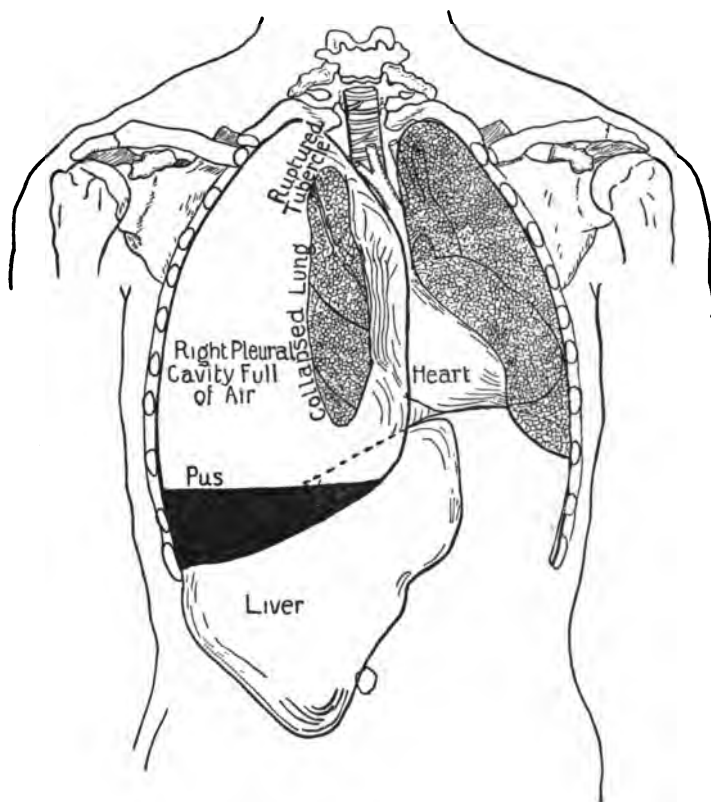


FIG. 108. A diagram of the chest showing pneumothorax on the right side. A small tubercle on the surface of the right upper lobe has ruptured, allowing air to enter the pleural cavity from the bronchi. The elastic lung collapsed like a toy balloon which has been pierced. There is no longer the normal negative pressure in the right pleural cavity, but the pressure there is equal to or above the atmospheric pressure. The result is that the heart is pushed (or sucked by the negative pressure) to the left side and the liver falls. The contents of the ruptured tubercle will set up a pleurisy and pus will collect in the pleural cavity. The condition is pyopneumothorax.

the hot-water bottle, or a mustard plaster—by the ice-bag, by painting the chest with iodine, and, lastly, by drugs.

TUBERCULOUS PERICARDITIS is one of the hardest conditions for even the doctor to recognize. When it is acute there is heard with each heart-beat a rubbing or scratching sound,

which may be very loud. Strange to say, this rub is usually painless, while the similar, but much fainter, pleural friction of pleurisy is very painful (Fig. 109).

The presence of fluid in the pericardial cavity is very hard

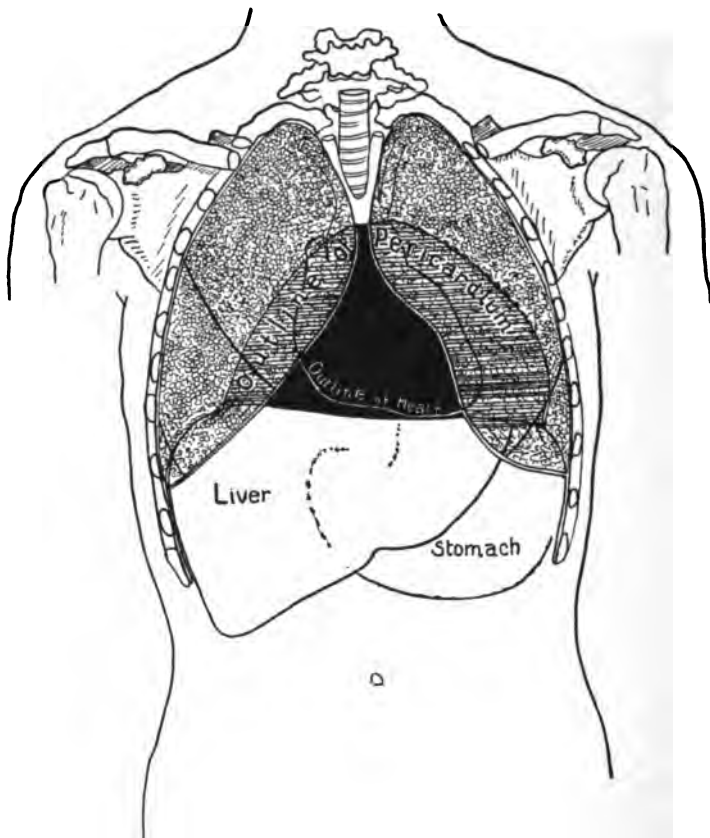


FIG. 109. Diagram of the chest of a case of pericarditis with effusion.

to determine, because we usually think it is in the pleural cavity; and, indeed, when it collects slowly, it may distend the pericardial sac so that this fills practically the whole left half of the chest. It may contain between one and two litres of fluid. The symptoms which the nurse might detect are the following: increasing dyspnoea, with the breathing free and

painless; dusky cyanosis; a pulse that is weak, and especially so during a deep inspiration—in fact the beats may entirely fail then (“pulsus paradoxus”). The fluid should, as soon as detected, be removed by tapping.

Adhesive pericarditis, or adherent pericardium, is the result of an acute inflammation of that sac. The two layers of serous membrane become bound together, sometimes over the entire heart, by adhesions. Then at each heart-beat the pericardium must move with the heart. Since normally the outer surface of the sac is but loosely attached to surrounding organs, this condition makes practically no trouble, unless the inflammation is severe enough to extend through the sac and make this tightly adherent to these surrounding organs. Then a very serious heart disease follows, yet one often difficult to recognize. When the external adhesions are strong, the work of the heart is greatly increased. The heart decreases considerably in size with each contraction, but now, in order to contract, it must pull in the ribs, pull on the pleura, and pull up the diaphragm along with the liver, which is attached to the lower surface of that muscle. The result of the greatly increased work is a huge heart, often more dilated than hypertrophied, and the symptoms are those of marked valvular disease or myocarditis. The treatment is the same.

TUBERCULOUS PERITONITIS.—This condition is very common, especially in the colored race and in children. The three forms mentioned in connection with pleurisy and pericarditis occur here also, but the acute form usually produces few, if any symptoms, and either the local condition is overlooked, or one of many wrong diagnoses is made, such as typhoid fever or a tumor of some abdominal organ. The latter mistake is not a bad one, since often even large tumors are present—masses of tuberculous matter, organs bound together, or fluid exudates tightly encapsulated in various parts of the abdomen. The ascites, or free fluids in the peritoneal cavity, may be extreme. On tapping it, which is done when there is much fluid there, from ten to twenty litres may be removed. There is usually much tympanites also (the distention of the bowels with gas), either diarrhoea or constipation, and an irregular fever. These cases do well after the simple operation of merely opening the abdomen, exposing the bowels to the air, and closing the abdomen again. Why

this should be so beneficial is not known, although theories are numerous enough; but of the fact there is no doubt.

The result of the chronic adhesive tuberculous peritonitis is remarkable. The adhesions of scar tissue bind the abdominal organs together and greatly distort them. That the bowels are not more frequently obstructed is remarkable.

In tuberculosis of the serous membranes one, two, three, or all of the sacs may be involved. But when more than one is involved (in this case the pleuræ being counted as one) a diagnosis of tuberculosis is quite safe. The only other disease which comes seriously into consideration is cancer. Other inflammatory diseases usually attack but one sac.

In the above paragraphs we have mentioned special treatments applicable to local conditions, but it is understood that the rigid treatment for tuberculosis of the lungs is always to be carried out for each of these conditions also.

TUBERCULOSIS OF BONES AND JOINTS is one of the commonest forms of tuberculosis in children. Practically all cases of hunchback ("Pott's disease"), of hip disease, of "white swelling" of the joints, and of "cold abscess," are due to tuberculosis. The disease begins, as a rule, in the bone just bordering the joint and progresses slowly, causing great destruction of bone and of the articular surfaces. The disease often ceases but it usually leaves the joint stiff.

TUBERCULOSIS OF THE KIDNEY is quite common. The tubercle bacillus may be carried there by the blood, but in most cases it travels up the ureter from the bladder (an "ascending infection;" tuberculosis of the lower genito-urinal tract is very common). The symptoms are those of pyelitis, and the result is often the entire destruction of the kidney. The treatment is to remove the organ.

SOLITARY TUBERCLE.—In the brain there sometimes forms a large mass called a "solitary tubercle," which may be as large as a hen's egg. It is a tumor and gives the symptoms of brain tumor.

TUBERCULOSIS OF THE ADRENAL GLANDS, called "Addison's disease," deserves especial mention. The adrenals are two small bodies, one just above each kidney. If they are really glands their secretion is an internal one—that is, it flows into the blood—for they have no duct. Just how these glands act normally we have no idea, but we know very well

what happens when they fail in their function. The person has attacks of exhaustion, which may come after exertion or spontaneously, when he feels so weak that he hates even to turn in bed. There may also be pain in the back under the short ribs, but this is not always present. The pulse becomes so weak that it can scarcely be felt. The person vomits incessantly without any relation to eating. At first these attacks occur at irregular intervals, but later the condition may be constant. At the same time, and usually this is the earliest symptom, the skin gets darker and darker, especially that of the face, hands, axillæ, nipples, waist line, groin, and dark spots appear in the mouth. The condition may progress for two or three years but always ends fatally.

Although this disease may be due to destruction of the adrenals from any cause, cancer for example, yet in the great majority of cases it is due to chronic tuberculosis of these organs.

Some think the symptoms are due to loss of the internal secretion of the gland; others, that they are really due to extension of the disease to the abdominal nervous system covering these glands. In favor of the first view is the effect of the drug "adrenalin," or "epinephrin," which is an extract of these glands and is now widely used. When applied to a mucous membrane, for instance, this drug causes marked contraction of the blood-vessels there, and hence it is of great value in checking hemorrhage. When injected into an animal it causes a marked rise of blood pressure.

No matter what the organ attacked, or what the form of the disease, the treatment of tuberculosis is practically always the same. It consists of the measures necessitated by the local nature of the disease, but in all cases the complete, rigid, open-air, forced-feeding treatment of consumption is to be conscientiously carried out, as described on page 266.

While the above are the most marked varieties of tubercular disease, yet any other organ may be attacked.

We now use the tuberculin test to aid in diagnosing this disease as well as in treating it, and in just the same way as we use it in cattle. Tuberculin is the poison that the tubercle bacillus forms. It sets this poison free in the fluid in which it is growing. What we use is this fluid after the germs have been removed by filtration. If a very small dose

of this filtrate is injected subcutaneously into a normal person, it should have no effect at all; but, if the person has anywhere in his body a focus of tuberculosis, there will usually be headache, malaise, and a rise of temperature during the next two days, usually in about eighteen hours. At the same time there should be a "local reaction"—that is, for a few hours the tuberculosis becomes more active. For instance, if the tuberculous trouble is in the knee, that joint should become more painful, more acutely swollen, etc. Tuberculin is also used as a remedy for this disease in patients without fever. If it is used for treatment, however, very small doses, so small that they cause no fever at all, are used, and at regular intervals. Some very good results are obtained in this way. This treatment is very similar to the Pasteur treatment for hydrophobia.

Diplococcus Intracellularis Meningitidis.—This germ is the cause of EPIDEMIC CEREBROSPINAL FEVER, also called "spotted fever." It is, as cocci run, a very tiny diplococcus (see Fig. 99), and it is so soon "swallowed" by leucocytes that we seldom see it outside of one; whence the name "intracellularis." The two cocci of the pair are rather flattened against each other.

There have been several bad epidemics of this disease in the world, and so many in this country that some authorities consider it an "American" disease. But large epidemics are rare. It often occurs sporadically, especially in country regions. Children are affected more frequently than adults. It seems not to be directly contagious, as it is seldom that two persons in the same family or house have it. It often seems to be a filth disease.

How it enters the body is not known. Some say that it enters through the nose, and the organism is in some patients so abundant there, that we carefully disinfect those secretions. Once in the body, the germ is probably carried about, and it chooses as its favorite place in which to settle and grow, the membranes which surround the brain. As you will remember, the brain is an exceedingly delicate organ and lies in a "water-bed." The fluid forming this bed lies between the dura mater—the outer, thick, strong membrane that lines the bony cavities of the skull and spinal canal, and the pia mater—a delicate membrane which immediately covers the brain

and cord, and in which tiny vessels run to the cortex. *Diplococcus intracellularis meningitidis* attacks the pia mater. The blood-vessels there become congested and pus collects, especially over the bottom of the brain and the back of the cord. The inflammation spreads to the inner walls of the brain, for this organ is really hollow, and its internal cavities—the ventricles—are full of the same fluid as that mentioned above. In very acute cases the pus will not have time to collect before the poison of the germ kills the patient (Fig. 110).

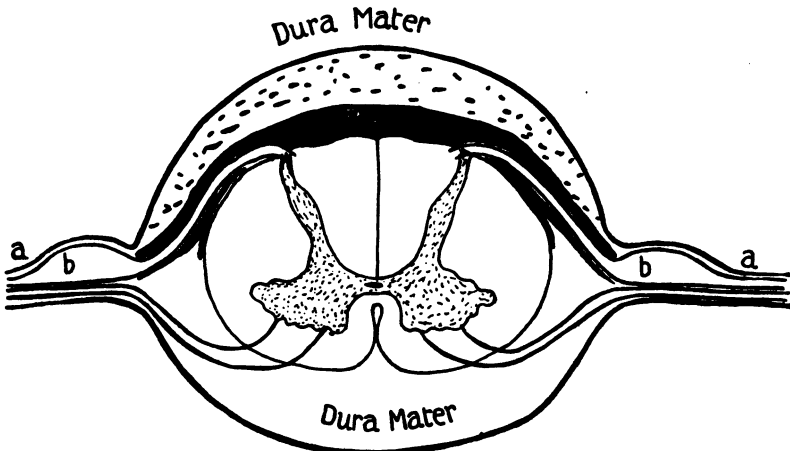


FIG. 110. A cross-section of the spinal cord from a case of epidemic cerebro-spinal meningitis. Note that there is an increased amount of cerebro-spinal fluid, and that the pus is chiefly posterior. If a hollow needle is stuck through the dura a cloudy fluid will gush out. a, spinal nerve; b, posterior root ganglia.

The symptoms of this disease are chiefly due to the direct effect of the poison of this germ on the cortex, partly to the pressure of the fluid, whose quantity is increased both around the brain and inside the ventricles, and lastly, to the settling of the germ in other organs, to which it is carried by the blood. The inflammation spreads also along the nerves as they leave the brain.

The onset is usually very sudden and is marked by a chill, vomiting, headache, fever, stiffness of the neck, and drowsiness. The patient is irritable and restless, delirious, sometimes maniacal. Later comes stupor and even coma. The neck is stiff; try to bend it, and you cannot do it. The headache is excruciating and comes in spasms. There are exquisite

pains in the back and limbs. Sometimes the stiffness of the neck extends to the back, and the whole body is a rigid rod. Sometimes there are convulsions. The inflammation creeps along the optic nerve, and the patient at first cannot endure any bright light—"photophobia"—and later may be blind; along the auditory nerve, and he becomes very sensitive to even slight sounds, and later becomes deaf; along the motor nerves of the eyeball muscles and the patient becomes cross-eyed or wall-eyed; along the facial nerve, and there is facial paralysis, etc. The fever is usually very irregular. Small subcutaneous hemorrhages—petechiæ—may appear under the skin, whence the name "spotted fever." Fever blisters are very common on the lips. The increased intracerebral pressure is shown by the very slow pulse and the Cheyne-Stokes respiration. There is usually a high leucocytosis.

During an epidemic, some cases are scarcely ill; some are very ill and die in a few hours; many die within five days. But most of the cases are ill for weeks or months, being now almost well, then very ill; relapse follows relapse; complication follows complication—pneumonia, arthritis, pleurisy, etc. Some patients entirely recover, but such are rare. The majority have some sequel to remind them, as deafness, blindness, facial paralysis, a paralysis of the eye muscle. Some have the distressing symptoms of chronically increased intraventricular pressure—severe paroxysmal headaches, vomiting, convulsions, and mental feebleness.

The diagnosis is made by a lumbar puncture—that is by sticking a long hollow needle through the back and into the spinal canal, and letting some of the cerebrospinal fluid escape. In this the germ can be found.

The treatment thus far is to make the patient comfortable, and to keep up his strength as well as possible by feeding and stimulation. The patient must be fed well, else he will waste to a skeleton. He is sometimes more comfortable in a warm bath, or with an ice-bag at his head. Lumbar puncture will often help the patient by removing some of the increased fluid.

Bacillus Influenzæ.—INFLUENZA or *la grippe* is sometimes (worse than epidemic) a truly pandemic disease. It is then an exceedingly contagious fever, and as such it has more than once swept with great rapidity over practically the whole civilized world, attacking even 41 per cent. of all exposed.

Yet we often see sporadic cases, and the specific germ—*Bacillus influenzae* (Fig. 100)—causing it is almost ubiquitous, being present in over 30 per cent. of the cases of chronic bronchitis, in cases of bronchiectasis, pulmonary tuberculosis, etc. The germ is everywhere, but conditions necessary for its rapid dissemination seem only occasional.

This bacillus is most apt to attack the respiratory tract. In doing this it causes acute coryza, and acute bronchitis with (and this is most important in the diagnosis) far more prostration and debility than are seen in other forms of bronchitis. For this reason it may resemble typhoid fever. There is much thin, greenish or bloody sputum. This is true "la grippe," but the name is applied to almost any severe cold, whether due to the influenza bacillus or not. The bacillus may cause acute pleurisy, also bronchopneumonia, or bronchiectasis. But it attacks many other organs besides those of respiration. In a second group of cases nervous features predominate—headache, profound depression, prostration, etc. In another group the gastro-intestinal tract is especially affected, and the patient suffers from nausea, vomiting, abdominal pain, diarrhoea, jaundice, collapse, etc. In a very important group there are no local signs, but the influenza may, with almost typical chills, simulate malaria, or may, with continued fever, simulate typhoid fever.

Among the various serious complications and sequelæ of influenza (and they are due to the same bacillus) are pericarditis, endocarditis, septicæmia, peritonitis, appendicitis (perhaps very commonly), nephritis, and often otitis media. It may cause nervous troubles of almost all descriptions—meningitis, abscess of the brain, paralysis, melancholia; it may even be followed by dementia.

That influenza seldom kills is indicated by the fact that its mortality is only 0.5 per cent. But as a disease which destroys health, few can equal it, as is shown by the foregoing list of sequelæ.

The diagnosis during an epidemic is easy, but that of a sporadic case is more difficult. The extreme nervous prostration it causes is very suggestive, but the unquestionable diagnosis is made only by the discovery of the germ itself; and, the more systematically this is looked for, the oftener and more unexpectedly is it found.

The treatment is, first of all, isolation, and disinfection of the secretions, especially the sputum. The patient must be kept flat in bed; this is the best way to prevent complications and sequelæ. He should receive the fullest diet possible and should be well purged and stimulated. The convalescence is long and tedious; it may take months, and for even years the patient may not be well. For this reason, a change of climate, when possible, is a great aid.

Bacillus Diphtheriæ.—**DIPHTHERIA.**—That diphtheria is caused by *Bacillus diphtheriæ* (Fig. 100) there is now no doubt. This germ lodges usually on the mucous membrane of the throat, multiplies there rapidly, kills the surface of this membrane, and causes an inflammation with the exudation of much fibrine. The dead mucous membrane and the fibrine form a white, leathery skin, or false membrane, which gives the name "diphtheria" (leather). Strip off this membrane and the bleeding surface of the submucous tissue is exposed. The germ forms also a virulent poison, or toxin, which is absorbed into the blood and carried over the body. This toxin causes the fever, pains, and all the other general symptoms of the disease. The germs themselves all remain in the membrane; only in the severest cases do they invade the blood. They are on the body, not in it.

The onset of symptoms in a case of diphtheria occurs usually from two to seven days after the exposure. It begins with fever, chilly feelings, pains in the back and limbs, headache, and general malaise. Next the patient notices that his throat is slightly sore, and a little patch of white membrane is seen, usually on a tonsil. The throat does not look very red, nor are the tonsils much swollen. The glands in the neck swell, and the face becomes ashen gray. The next day one notices that the patch of membrane has enlarged and has crept beyond the tonsil. Then one is sure of the diagnosis. This membrane may grow rapidly, extending over the soft palate to the posterior wall of the pharynx, up into the nose, down to the larynx and trachea, and even into the fine bronchi. It may extend along the Eustachian tube to the middle ear, along the nose into the nasal sinuses, down the œsophagus into the stomach. The membrane is white, thin and delicate, when small in extent, or yellow, and tough when thick. The tissue below it may be very necrotic and

slough deeply. In the ordinary case the fever is never high and soon falls to normal. The throat is not very sore, and the patient doesn't feel very ill—not nearly so ill as in acute tonsillitis. This is really unfortunate, for diphtheria is a far more serious illness. After about ten days the membrane loosens, and falls off in shreds.

Bacteriological examination is necessary for diagnosis, since some cases really cannot be told on inspection alone from acute tonsillitis, and other cases have no membrane at all. A culture is easily taken, and a report can be made in twelve hours. Again, other germs, especially *Streptococcus pyogenes*, can cause a membrane very similar to that of diphtheria, and cases with such a membrane are very much less contagious than diphtheria.

Some patients are terribly ill from the extreme toxæmia. Of these, some have little membrane, some much; some have no fever, even a subnormal temperature until death. Other cases at no time feel at all ill, and can be isolated with difficulty. In the throats and noses of many cases whose fever has subsided, and all of whose symptoms have disappeared, the virulent germs live for weeks. Such persons are very dangerous to others. Those with nasal diphtheria are especially dangerous, for while some of them feel ill, the majority have no symptoms at all, not even fever, and harbor the germ for months. They especially spread the disease to others, some of whom may be more susceptible to that germ than they, who die of diphtheria of the most virulent types.

One of the worst forms of diphtheria in children is that which attacks the larynx and is called "membranous croup." The child may have a rough, croupy cough for a day or two, then suddenly almost suffocate. He tosses about, sits up in bed, and struggles to draw his breath. He becomes quite blue. Severe cases, if not relieved by tracheotomy or intubation, will suffocate. In milder cases the paroxysms are soon over but may recur later.

Bacillus diphtheriæ can cause, in addition to diphtheria of the mucous membranes, a membrane on the skin around recent wounds; and diphtheria of the eye is a serious condition.

Diphtheria is a disease always present, but it often occurs in severe epidemics, and it is of especial virulence in the country regions. Perhaps the germ is spread chiefly by mild

cases and convalescents. It is spread by particles of the membrane on clothes, etc. The germ has wonderful endurance and can live in clothes or toys, etc., for even five months. It is also carried in milk, where it grows without making it sour, or in any appreciable way altering it. It attacks children for the most part, although adults—nurses and doctors especially—are often affected.

Among the serious complications of diphtheria are the following: acute toxic myocarditis, the common cause of the sudden death of diphtheria patients, severe nephritis, due to the diphtheria toxin; and bronchopneumonia, due to an extension of the inflammation from throat to lung, and a common cause of death. Many persons tell of coughing up "tubes" from the lungs. These tubes are the lining of diphtheritic membrane from the larger bronchi. *Streptococcus pyogenes* is an organism which seems to flourish when in company with *Bacillus diphtheriæ*, and many severe inflammations in diphtheritic cases are due to the former organism.

Among the important sequelæ are various paralyses, which often develop during the second and third weeks of convalescence, especially those of the throat and eye muscles, and, less often, of the limbs. These occur in almost one-fifth of the cases. The diphtheria may have been so slight that the patient doesn't know he has a sore throat until the paralysis of the throat begins, when his voice becomes nasal and he begins to regurgitate liquids through his nose.

Treatment.—Since the manufacture of diphtheria antitoxin the dread of diphtheria has to a high degree disappeared. Formerly it was a very serious disease, which killed almost half of those whom it attacked; but during the past few years the mortality has fallen from 45 to 25 per cent., then to 15 per cent., and recently to 10 per cent. When the use of antitoxin is still more common, the mortality will drop even more. This enormous saving of children's lives, of whole families even, can scarcely be appreciated.

A germ is dangerous because of the poison, or toxin, that it produces. The diphtheria bacillus, for instance, is scarcely ever inside the body. It first forms the membrane on the wall of the throat. Here it multiplies and produces a poison, or toxin, which soaks into the blood and produces all the symptoms of the disease. As soon as this poison, or toxin, gets into

the blood, the body at once begins to manufacture an antidote for it. When enough antitoxin has been manufactured, the person begins to get well, and his blood is rich in antitoxin. But it happens in about half the cases that the toxin kills the patient before his body has produced sufficient antitoxin. Now it makes little difference from whence this antitoxin comes. The antitoxin from a person or animal that has recovered from diphtheria will do as well for our patient as antitoxin produced by his own body, if we inject it into him. So the method is as follows: A horse is made ill by injecting into him a dose of diphtheria toxin. The bacilli are not injected, for accurate dosage would be impossible if we used live organisms, as these could multiply and kill the animal. But fortunately the bacilli pour their toxin into the fluid in which they are growing, and by filtering this fluid free from bacilli, we get its toxin and so can give accurate doses. A dose estimated as being just short of fatal is given. The animal becomes very ill, but recovers, having manufactured antitoxin enough to neutralize a dose just about as large as the one given. Give it another dose of the same size and no illness will follow; but double the dose and the horse will be as ill as before, and, when it recovers, its blood will have an antitoxin potent enough to handle two of the original doses. The next time three doses of toxin must be given, then four, then five, and so on until the animal is able to stand a thousand or more of the original doses without being ill. Its blood-serum will then contain antitoxin of great potency, and a little of this injected into our patient's body would greatly help out by supplying "ready made" a large quantity of such antitoxin as his body is trying to manufacture. So the horse is bled, and the serum put in little bottles, one dose in each. On every bottle is a label stating the number of "units" (500, 1000 3000, etc.), that its contents contain. What "500 units" means we have not space enough to explain, but it refers to the potency of that particular quantity of antitoxin, as determined by experiments on guinea pigs.

In an ordinary case we inject about 6000 units of antitoxin at once and repeat the injection at intervals until we see the membrane shrivel up and loosen. If there is a membrane on the larynx, 16,000 units are injected at one dose. If the child, even a tiny baby, is very ill, even 100,000 units

may be given in the course of a day. The antitoxin itself does very little harm. It may make some joints very painful, or cause a severe skin eruption, but these are not serious troubles, and are trifles compared with the danger from the diphtheria.

If there is danger of suffocation, a tube for the child to breathe through is put into the larynx, ("intubation"), or a hole is cut into the trachea just below the larynx ("tracheotomy"). The child should be nursed in a cool, well ventilated room. If there is difficulty in breathing, the air should be saturated with moisture, which can be accomplished by means of a steam kettle designed for that purpose. The patient must rest quietly in bed, even into the convalescence, and the heart should be guarded against sudden movements. Liquid diet is given, and as much water as it is possible for the patient to drink.

The local treatment should be thorough and regular. Swabs, gargles, etc., are given regularly. In giving them the nurse must be very careful that the child does not cough some of the membrane into her face. Those exposed to diphtheria should take an occasional small dose of antitoxin, perhaps 500 units, as a prophylactic measure. All cases of diphtheria, however slight, and old cases in whose throats or noses the diphtheria bacillus can still be found, no matter how long after the acute attack, should be isolated, for they can spread the disease.

Shiga's Bacillus.—BACILLARY DYSENTERY.—Only very recently was the germ of this disease discovered. Formerly the diagnosis for adults was "amœbic dysentery," for children "summer diarrhoea." It is an acute infection caused by bacilli. It occurs especially in the tropics, but practically everywhere, and particularly in summer. It is very contagious and may occur in terrible epidemics; it is the scourge of armies. The chief symptoms are the passage of frequent scanty stools of blood and pus, with considerable pain and fever. While there are no real ulcers in the bowel, the mucosa in a severe case is necrotic over large areas; indeed, the whole colon wall may be practically killed by the toxin of these bacilli.

The onset is usually sudden, with fever, abdominal pain, and the passage of small amounts of blood and mucus. There

is constant desire to defecate, and great straining during the attempt. The severity of the symptoms, the prostration, and the toxæmia increase, and death may occur in a few days. The milder cases last two or three weeks, and the subacute cases for months.

Since we do not know how the bacillus of this disease enters the body, intelligent prophylaxis is impossible, but the evidence seems to show that water is the source, and that the same precautions should be used as in typhoid fever. The milder cases are self-limiting, with a course of eight or nine days.

The diet should be milk and broth, and the stools should be examined for curds. If these appear, other liquids must be substituted. Rectal irrigations are the best treatment, of water at 100°, with alum, or lead acetate, or, best of all, silver nitrate. The rectum is very sensitive and irritable, and must be anæsthetized before the irrigation by a cocaine suppository. The patient lies with his hips elevated on a pillow. Many drugs are used,—Epsom salts, laudanum, ipecacuanha, corrosive sublimate, and bismuth. Morphia is the only drug that will quiet the pain and tenesmus.

Bacillus Lepreæ.—LEPROSY is a chronic infectious disease caused by *Bacillus lepræ*. As in tuberculosis, the characteristic lesions of this disease are small tumors, which develop in the skin and along the nerves.

Leprosy is common in Scandinavia, Iceland, and the Sandwich Islands; in Asia it is very common. There are about 500 cases in America. It is communicated by direct contagion, but it is not very contagious. It is probably not inherited. The fear of it in Eastern countries is explained by the fact that under the head of leprosy Orientals group many skin diseases.

There are two varieties. The "tubercular variety" is characterized by the presence of pigmented (later white) spots in the skin and the formation there of little nodules, which later ulcerate, and then heal, leaving a scar. These lumps, open sores, and contracting scars give the patient a hideous appearance. The hair, eyebrows, and eyelashes fall out. The fingers and toes may ulcerate and drop off. The sight is soon lost.

In the "anæsthetic variety" the tumors grow in the nerves,

and the tissues which supply these become numb and atrophy. Patients with this variety live for years without showing any conspicuous signs of the disease.

There is practically no treatment, except to dress the open ulcers. The patient should be well isolated.

Bacillus Mallei.—GLANDERS is a disease from which horses suffer, and which is caused by a specific germ, *Bacillus mallei*. These bacilli cause the formation of a multitude of little tumors. When these are in the nostrils, the condition is called "glanders." When they lie under the skin, it is called "farcy." Man gets this disease from a horse by accidental infection. The nodules in the patient's nose rapidly ulcerate, and from these ulcers is discharged considerable offensive nasal matter. Pneumonia often follows. Acute glanders may be fatal in eight or ten days; chronic glanders may last for months with only the symptoms of a chronic coryza. In acute farcy in man, the many "farcy buds"—nodules—in the skin soon suppurate, leaving ulcers. Abscesses form in the muscles also and in the joints. The acute form of farcy is practically always fatal; very chronic cases may get well.

Bacillus Tetani.—TETANUS or "lockjaw" is an all too common malady and is due to a specific germ, *Bacillus tetani* (Fig. 100). This bacillus is a normal inhabitant of the intestines of cattle, and hence it is always present in manure and in the earth. There is *always* great danger of its following wounds made by dirty instruments, especially those received in a stable. In some localities it kills off over half the newborn children because proper cleanliness is not used in caring for the navel. Accidental infections with this germ are much to be feared when inoculations, vaccinations, etc., are made carelessly. Also this is the germ that makes celebrations on the Fourth of July such serious matters.

The tetanus bacillus stays in the wound; it does not, like most germs, invade the blood and organs. But in the wound it produces a toxin 200 times as poisonous as strychnine, and this toxin is responsible for all the symptoms of the disease. These symptoms do not appear until two days or more after the injury. Those first noted are a stiffness of the neck and difficulty in moving the lower jaw. This stiffness increases and extends over the muscles of the body until the jaw is locked and, during the spasms, the whole body is rigid.

The patient's back is held stiffly in a convex position, so that he may, instead of lying flat, rest, arched, on head and heels. The patient is as if held in a vise—unable to move a muscle, to speak, or even to breathe, and he may, therefore, suffocate. While this extreme rigidity is present only during the paroxysms, which are of varying duration, yet between these the body is not perfectly relaxed. These spasms are brought on by even slight stimuli, like a noise or a touch. Over 80 per cent. of such patients die, and by far the majority of these within four days.

In treatment, the care of the wound is most important, for here are all the germs, and here all the poison is manufactured. This should be opened and thoroughly cleaned out. The patient is kept in a dark room, and as quiet and little disturbed as possible. During the paroxysms morphia and chloroform should not be spared. An antitoxin similar in character to the diphtheria antitoxin is now used and is much more effective in preventing the disease than in curing it after it has developed. Every patient who comes into our hospital dispensary with a dirty wound receives a dose of this antitoxin.

Spirillum Cholerae Asiaticæ.—ASIATIC CHOLERA is a disease caused by a specific germ, *Spirillum cholerae asiaticæ* (Fig. 101). Cholera is endemic in India and has been epidemic in other countries, including our own. Its most important symptoms are severe diarrhœa and collapse.

The germ enters through the mouth and reaches the intestine where it multiplies enormously, but it does not invade the body. The symptoms are caused by its toxin, which is absorbed through the intestinal wall. The fact that cholera is not highly contagious is indicated by the fact that nurses and doctors are seldom attacked by it.

This disease is spread almost always by the stools of a person afflicted with cholera. These infect drinking water. Perhaps flies also aid, since they feed on these stools and then infect our food. Those handling the soiled linen of cholera patients often contract the disease.

Cholera usually begins with mild diarrhœa, colic, and considerable malaise. These symptoms last a day or two, and then comes the "stage of collapse." The diarrhœa becomes profuse, with incessant vomiting, and is followed by

collapse. The blood seems to pour all its water into the intestine to wash out the germs, and because of this loss of water, the body visibly shrinks, the thirst is excessive, the cheeks become hollow, the eyeballs become sunken, the skin over the body wrinkles, the secretion of urine stops, and the pulse becomes very weak. Although the temperature is high, the skin feels cold and clammy. Its general color is ashy, but the hands and feet are blue. The cramps in the muscles are terrible. The intestines are soon washed clear of any fecal matter and bile, and after that the profuse stools and vomitus consist of water and salts from the blood, and in them are many little white particles of mucus and intestinal epithelium. From their appearance, these movements get the name of "rice-water" stools.

The stage of collapse lasts from two to twenty-four hours, and after this, if the patient survives, the "stage of reaction" begins. The diarrhoea diminishes, the warmth of the body returns, the urine is again secreted, and the patient gradually recovers.

Individual variations in susceptibility are great. Probably only a few of those exposed to cholera during an epidemic really catch the disease, and among those who do contract it, all degrees of severity occur. Some have only a mild diarrhoea for a day or so—nothing more; while those at the other extreme die before the diarrhoea even begins. The mortality of the various cholera epidemics varies from 30 per cent. to 80 per cent.

The prophylaxis consists in isolation and in disinfecting the stools, vomitus, and bed linen; the treatment, in keeping up the patient's strength by stimulants (hypodermic), in warm baths, in washing out of the bowels by large enemata, and in supplying the blood with water by the subcutaneous injections of large quantities of the proper fluids.

Bacillus Pestis Bubonicæ.—BUBONIC PLAGUE or "the plague" is caused by a specific germ, *Bacillus pestis bubonicæ* (Fig. 100). The characteristic features of this disease are the swollen inflamed lymph-glands, called "buboes," carbuncles, pneumonia, and, often, hemorrhages.

This terrible epidemic, "black death," the most fatal of all acute diseases (it has a mortality of 80 per cent. to 90 per cent.) once swept over Europe, killing a quarter of the

population, and even now is killing in India nearly a million persons a year. An occasional case reaches Northern seaports. It is considered a filth disease and does confine itself chiefly, although not entirely, to the crowded, filthy quarters of a city. One person catches it from another but (what is very important) the germs are spread by fleas and rats, and the problem of preventing epidemics of this disease is the problem of destroying rats. Yet the plague is not so very contagious; it is contracted by few nurses, doctors, or soldiers on guard in the infected quarters.

At the onset, the patient suffers from "headache, backache, stiffness of the limbs, a feeling of anxiety and restlessness, and great depression of spirits." The temperature rises for three or four days, drops slightly, then rises again—to a higher point than before. Fatal cases usually die in extreme collapse during this secondary fever.

The "buboes," or swollen glands, appear between the third and fifth day. They may disappear, but as a rule they suppurate, and the abscesses break through the skin. Hemorrhages under the skin are common. These last were the "plague spots," or "tokens of the disease," and gave it the name "Black Death."

Other cases, before the buboes appear, die from septicæmia caused by the plague germs, while the most fatal form of the disease is a pneumonia due to this germ (mortality 96.6 per cent.).

The sputum, urine and stools may contain vast numbers of *Bacillus pestis bubonicæ*. The diagnosis is not difficult, because these germs are easy to recognize.

In treatment the most rigid isolation must be preserved. All evacuations must be destroyed, and all clothes disinfected. The patient should be made comfortable, and proper local treatment (ice and compresses, surgical measures, etc.), should be applied to the buboes. The dead should be cremated.

Vaccination measures now in use promise to prevent the spread of the plague epidemics, while in some countries a successful war of extermination of rats is in progress.

Bacillus Anthracis.—ANTHRAX.—Anthrax is the most widespread and fatal of all animal plagues. It is caused by a germ *Bacillus anthracis* (Fig. 100), which attacks the sheep

and cattle of almost every country, but especially of Asia, Russia, and France. If a field has become "infected" by this germ (from the bodies of animals killed by this disease) there is for years danger that other animals grazing there will contract the disease.

Farmers, butchers, and those handling the wool, hides, or flesh of infected animals not seldom contract it. If this bacillus gets into a scratch on the skin, it causes a "malignant pustule," a very bad form of boil. This boil has a black centre, since there the flesh is dead, and a much swollen periphery. If the whole boil is not quickly cut out, blood-poisoning may, and in about a quarter of the cases does, follow, and then the person dies in a few days. Or the germ may enter the body in the meat or milk of diseased animals and cause a rapidly fatal enteritis; or it may be inhaled with dust and cause a fatal bronchitis, called "wool sorter's disease," or "rag picker's disease." So virulent are these diseases that the patient sometimes dies in a few hours from the onset.

Spirochæte Pallidum.—SYPHILIS.—This disease and the one next to be described cause, without doubt, more suffering, physical and mental, than all other diseases put together.

Syphilis, "lues," or the "bad disorder" is caused by a germ, *Spirochæte pallidum* (Fig. 101). This germ produces at the point where it enters the body, often after even four weeks' delay, a hard, indolent sore, which is sometimes so small that it is overlooked by the patient, and which soon ulcerates. It heals slowly, leaving a permanent scar. This is the "initial lesion," or "primary sore," and is called a "hard chancre."

From six to twelve weeks after the appearance of this sore begins the secondary stage, or stage of general infection of the whole body. The patient has a fever, becomes anæmic, complains of rheumatic pains in the bones. These pains are worse at night; there is a chronic sore throat; the hair falls out; often the eye inflames. The most important feature, however, is a skin rash, most marked as a rule over the trunk. This occurs in such a variety of forms that description would be useless. In the mouth and on the tongue are seen white spots called "mucous patches." The patient seldom feels very ill during this second stage and is almost never in bed. This is the stage when the disease is very

easily communicated to others. The whole skin, while the rash is present, may be infectious, and the saliva is especially dangerous.

After the secondary stage—sometimes at once, often years later—begins the tertiary stage, which is characterized by the formation of tumors called “gummata.” These may develop in any organ of the body. Tertiary lues can simulate almost every disease of almost any organ, and if left without treatment may equal the worst in its distinctive effects. The blood-vessels, brain, and liver suffer worst.

Years later locomotor ataxia or general paresis may end the sad picture.

Among these lesions, the chancre, the mucous patches, and some of the skin rashes are very contagious, and at the point where they infect another person the chancre appears. As syphilis is supposed to be spread only by sexual irregularities, it is well to explain the fact that perhaps in 10 per cent. of the cases the infection is purely accidental. Nurses and doctors catch syphilis while treating cases, and hence have the sore on hand or finger; it is often on the lips, transmitted by kissing, or by drinking cups, pipes, or dirty towels. Sometimes the disease occurs in epidemic form, and once, in 1494, it was pandemic.

The children of luetic parents are born dead or die within six months. If such children survive, they bear throughout life some marks of their inheritance.

In the case of syphilitic patients great caution must be observed, but one should remember that the vast majority of cases in women and children are accidental infections, and that by no means all men are to be blamed for their trouble. The disease is easily caught, and nurses especially, who must bathe their patients, dress the patients' ulcers, etc., should be alert to their own danger.

The medicinal treatment is very satisfactory, if continued for two full years, even when all symptoms have disappeared in two weeks (a hard task for most men). The dishes, the linen, everything, in fact, which the patient touches should be as thoroughly isolated as those used in smallpox cases. During the time mercury is given, a potassium chlorate mouth wash should be used to prevent salivation,—that is, sore gums and an increased flow of saliva.

Micrococcus Gonorrhœæ.—GONORRHOÆAL INFECTION.—The *gonococcus* (Fig. 99) is one of the most important and destructive of germs. Gonorrhœal urethritis, the local trouble which it causes, may seem trifling, but this is never the case. In males it may remain latent for many years. The urethritis seems cured, but the germ is alive, and many think that it can never be eradicated. It often causes "gleet," stricture of the urethra, prostatitis, and is the commonest cause of sterility. It invades the blood and organs. Some of the worst cases of heart disease and a very common form of chronic rheumatism are due to it. It may cause also "blood-poisoning," pericarditis, peritonitis, various abscesses, and many other conditions. This organism causes over three-quarters of the cases admitted to the wards for female troubles, and most of these must be operated on. It also explains many more than half the cases of blindness of infants.

Spirochæte Obermeieri.—RELAPSING FEVER.—This fever is caused by a germ *Spirochæte Obermeieri* (Fig. 101), a minute, spirally coiled, thread-like organism, which during the fever can be seen moving actively among the red blood-corpuscles. It occurs in India especially, but cases have been found in this country. It is contagious and occurs especially among the very poor, who live in overcrowded hovels, and are almost starving, hence the name "famine fever."

The fever occurs as a series of sharp attacks, whence the name "relapsing fever," each lasting six or seven days, and separated by intermissions of about the same length of time. The patient may have from two to four or more such relapses. The fever of these attacks begins suddenly, often with a chill; the temperature is very high and falls by crisis. There are also nausea, vomiting, and pain in the back. The disease is rarely fatal.

Streptococcus Pyogenes.—ERYSIPELAS.—Erysipelas is an acute infection of the skin, due to *Streptococcus pyogenes* (Fig. 99), and characterized by a diffuse inflammation with very little pus formation. It sometimes occurs in epidemics, especially in the spring, but in olden times the local epidemics in hospitals made those institutions about as dangerous places as possible for a patient. It is really not very easily communicated to a healthy person, but the danger is great if the person exposed has a flesh wound or is a woman just after labor. For

the latter it is a very serious matter. The form occurring about the navel of the newborn child is fatal. The germs are carried about the body by the blood and cause abscesses in the lung, spleen, kidney, etc., and the worst forms of endocarditis.

In the "idiopathic form" the face especially is affected. The period of incubation lasts from three to seven days. The attack begins with fever, and often with a chill. The flush appears first on the bridge of the nose, and spreads over the cheeks, head, and neck. The skin becomes red, hot, smooth, and oedematous. The margin has a well-defined raised edge. Blebs often form on the skin, and, in severe cases, abscesses. The eyes may be closed by the swelling; the lips may become huge. The temperature is high for four or five days, and then often falls by crisis. The process may spread upon the trunk, covering almost the entire body. Among healthy persons the mortality is only from 4 per cent. to 7 per cent., but among drunkards and old persons the result of the toxæmia is often death.

Erysipelas is a "self-limiting" disease, and its course cannot be shortened. One prescribes nutritious diet, drugs to give comfort, good tonics; while local applications to the skin of cold water, of weak antiseptics, or of ichthyol add much to the patient's comfort.

Micrococcus Melitensis.—MALTA FEVER.—Malta fever is a noncontagious disease caused by a specific germ, *Micrococcus melitensis*, and characterized by the undulatory course of the fever. Other names, derived from the regions where it is most common, are "Neapolitan fever," "Rock fever" (Gibraltar), and "Mediterranean fever."

Its course lasts six months or more and consists of a series of febrile attacks, each lasting from one to three weeks, and separated by a few days of apyrexia. With fever there are definite inflammation of joints and profuse sweats. The mortality is only about 2 per cent.

CHAPTER XIII

OTHER DISEASES CAUSED BY BACTERIA

Tonsils.—The tonsils are two almond-shaped bodies, one on either side of the pharynx at the point where mouth and throat meet. They are really lymph-glands. In addition to the two large tonsils are many tiny ones, which form a ring around the orifice of the throat. This ring covers the base of the tongue, extends up the pillars of the fauces, and crosses the roof of the pharynx. The tonsils in this situation are called adenoids (Fig. 46). During childhood the tonsils are large, but they diminish in size with age, until they can scarcely be seen. While we do not know their exact function, they seem to be protective organs.

ACUTE FOLLICULAR TONSILLITIS is an inflammation of the tonsils caused by *Streptococcus pyogenes*. On the surface of the normal tonsil are several little depressions, which are the mouths of the little pockets or "crypts" of the tonsil. In a case of tonsillitis, it is in these crypts especially that germs flourish and form little abscesses, whence the name "follicular tonsillitis." The tonsils, usually both, are much swollen, and the infected crypts can be seen as little white spots, one or many on each tonsil. A little stream of pus is often seen trickling down from the crypts.

An attack of tonsillitis is usually the result of exposure to wet or cold. It often begins with a chill. The fever for a few days is high. The patient suffers from headache, much malaise, pain in the joints, etc.; he finds on swallowing that his throat is very sore; the glands at the angle of the jaw are swollen, and sore to the touch. There is scarcely a disease which makes one feel so wretched. The attack lasts about a week.

The other tonsils, including the adenoids, may suffer in a similar way. Those low in the throat—at the root of the tongue—when inflamed give intense pain when the patient swallows.

Tonsillitis is a disease of young persons especially, but also of adults. It occurs in epidemics, especially in crowded institutions, and most often in the early spring and fall. Attacks tend to recur.

A good dose of calomel should be given at once. An ice-bag or cold compresses on the throat will give great relief. There is great difference of opinion, but some of the best authorities condemn the use of gargles, swabs, and other local treatment. The patients find great relief in sucking ice and should drink large quantities of cold water. Salicylic acid, aconite, guaiacum, and Dover's powders are useful. These patients will take only cold nourishment—many only ice-cream.

Suppurative Tonsillitis.—In follicular tonsillitis the little abscesses are superficial and open into the mouth, but in suppurative tonsillitis, or "quinsy," they are so deep in the tonsil that they cannot discharge externally, and hence the pus collects as a large abscess in or underneath the tonsil. This may push the tonsil and pharyngeal wall to or even past the mid line of the throat, and when both tonsils are affected they practically close the throat. The temperature is high, the prostration is extreme. So far as symptoms go, there is scarcely a more distressing disease. If let alone the abscess will in time "come to a head" and rupture, usually into the mouth; but it is better to lance it early, for otherwise we prolong the intense misery of the patient, and a fatal result may follow. In addition to this simple operation all the treatment for ordinary tonsillitis should be given.

CHRONIC TONSILLITIS.—Many persons have chronically enlarged tonsils. In some this condition has existed since birth, while in others it is the result of an acute infectious fever, such as scarlet fever, diphtheria, or acute tonsillitis. Again, it may be part of a general enlargement of all the lymphatic structures of the body,—lymph-glands, thymus, spleen, bone-marrow, etc. There is a rare and interesting condition known as "lymphatism," or "status lymphaticus," in which this general enlargement of lymphatic organs is the chief symptom. Patients thus affected are especially susceptible to acute-infections and to slight injuries, and are very liable to sudden inexplicable death. Anæsthetics are never given to a person who has even the slightest trace of

this condition. As a rule, however, only the tonsils, including the pharyngeal tonsils, or adenoids, are enlarged in cases of chronic tonsillitis. The effect of adenoids on the mechanics of respiration is discussed on page 75. Here we shall speak only of the infections of these structures and of the results of such infections. It is important to remember that tonsils which are not at all painful, and which on inspection do not look swollen or inflamed, may be the seat of a chronic infection and a source of trouble for the whole body. To the germs which they harbor and which are constantly entering the blood, are due repeated attacks of acute articular rheumatism, many cases of chronic rheumatism, perhaps the majority of cases of acute endocarditis, many cases of St. Vitus's dance, many cases of acute nephritis, and numerous eye and ear troubles. These chronically inflamed glands are also a favorable soil for the tubercle bacillus to multiply in, and there is good reason to believe that in many cases of tuberculosis of the glands of the neck ("scrofula") and of the apexes of the lung the germ has entered at this portal. Cases of chronic tonsillitis are very liable to diphtheria and to recurring attacks of acute follicular tonsillitis.

The importance of removing chronically enlarged tonsils is now fully realized. They should be cut out, not clipped off. This operation takes a little time and is rather disagreeable, but is not really painful. Adenoids should be removed as soon as discovered.

Rheumatism.—**ACUTE RHEUMATIC FEVER.**—That this acute, non-contagious fever is caused by some germ or its toxin is the opinion of most authorities, for it resembles closely those diseases which are undoubtedly infectious. But as to what that germ is few agree. It is certain that the attacks of rheumatism bear a definite relation to acute tonsillitis and to acute endocarditis, both of which are due to germs.

"Acute rheumatic fever" is an acute inflammation which attacks joints, and which on subsiding leaves them normal. It is a disease of temperate climates, and occurs in over half the cases during the first four months of the year. It attacks persons of all ages, but especially those from fifteen to twenty-five years old.

The attack, as a rule, begins abruptly. The temperature rises rapidly as the joints become swollen. Within one day

the disease is well developed. It is usually a polyarthritis,—that is, more than one joint is affected. The joints become swollen, red, hot, and exquisitely painful. The patient cannot move them in the least. Often the slightest weight of bedclothes is unbearable, and the patient cries out, for fear the visitor may touch him, jar the bed, etc. The joints most often involved are the knee, ankle, shoulder, wrist, elbow, hip, hand, and foot. The joints are not all inflamed at once, but the disease travels from joint to joint, one joint swelling as its neighbor subsides. The joints recover completely, no stiffness remaining. The prostration is extreme. Many patients have profuse acid sweats with a sour odor. The convalescence is slow. This disease is very apt to recur.

The complications and sequelæ of acute rheumatic fever are more important than is the disease. Rheumatism seldom kills a man (the mortality is less than 3%), but its sequelæ fill a large number of our hospital beds. To use Dr. Osler's expression, "rheumatism kills at long range." Its most important complication, especially during childhood, is acute endocarditis. The first attack of rheumatism leaves over half the cases with heart disease; the second, over 60%; and the third, over 70%. The endocarditis is of the simple variety that makes little or no trouble at first, but serious trouble years later. Tonsillitis was formerly considered a complication, but now it is recognized as the primary factor, and the present conviction of clinicians is that, were the throat properly treated, the number of cases of rheumatism, heart disease, and chorea would be greatly diminished. Pericarditis oftener follows rheumatism than it does any other acute disease. St. Vitus's dance is a common complication.

The treatment is to make the patient comfortable and prevent complications. Acute rheumatism is a self-limiting disease, and probably no drug will shorten its course by one day. Of course the throat should be treated, and the tonsils, even if only slightly enlarged, removed. The bed should be as comfortable as possible. The patient should lie between blankets and should, on account of the sweats, wear a flannel garment.

The diet should be "an alkaline diet," consisting chiefly of milk diluted with alkaline waters; hence meat is excluded. (Some think the whole trouble is an acid intoxication.) Large

amounts of water should be drunk. The pain in the joints may be relieved by very hot or very cold compresses, the Paquelin cautery, various liniments, or fixation by splints. Salicylic acid is the best drug to relieve the pain. It is given in large enough doses to accomplish this and then stopped, for it does injure the patient. Small doses are of no benefit; large doses sometimes cause mental symptoms.

The treatment during convalescence is most important; many cases of heart disease could doubtless have been prevented had the patient not got up too soon. He should stay in bed six weeks, but it is a difficult matter to keep children there so long.

SUBACUTE RHEUMATISM.—Many cases of this disease run a very mild or "subacute" course, and the patients, especially children, object to staying in bed. The inflammation of the joints in these cases is so slight as almost to be overlooked, but there is about the same danger of complications as in severe cases; hence the treatment is the same.

Almost any illness characterized by painful joints is spoken of as "rheumatism," but the term "acute rheumatic fever" is now limited to cases which answer the above description, and other cases are spoken of as arthritis. It is doubtful whether any so sharp line can be drawn.

SECONDARY ARTHRITIS, OR INFECTIOUS ARTHRITIS, is the name applied to a large group of cases in which the organism causing the trouble can be found in the joint. Arthritis can be a complication of almost any infectious disease, but the germs most frequently causing it are *Bacillus tuberculosis*, *Streptococcus pyogenes*, *Staphylococcus pyogenes*, the gonococcus, and the meningococcus. Cases of secondary arthritis may resemble acute rheumatism in their onset and course, and in the fact that they may leave many of the inflamed joints clear; but, as a rule, the germs make one or more joints their permanent habitat and cause a certain amount of permanent injury or even an abscess in the joint.

The treatment of these cases depends on the germ and is often operative. A little of the joint fluid should be removed with a syringe, and cultures made to determine the germ. If the fluid is cloudy,—that is, purulent,—the joint should be opened and the fluid emptied. A tuberculous joint is kept perfectly quiet, and the patient given the full treatment

for consumption. Other cases improve under hot-air treatment. If the joint shows signs of stiffening, massage, forced movements, and exercises may check this; but if these fail, one must prevent ankylosis in an unfortunate position. If the knee becomes stiff it should be fully extended, the elbow or ankle, bent to a right angle.

CHRONIC RHEUMATISM is the name given to a condition marked by chronic stiffness and pain and even some swelling in the joints. The pain is worse during bad weather. This condition occurs especially in elderly persons and those whose work exposes them to cold and damp; hence it is very common among the poor. Whether it is really related to acute rheumatism or not is doubtful.

The best treatment is massage, forced motion, and the Paquelin cautery; among drugs, aspirin and potassium iodide are especially useful. Change of climate and a stay at the various watering-places are very beneficial.

ARTHRITIS DEFORMANS.—This terrible disease, also called "rheumatoid arthritis," although it may not be at all related to rheumatism, seems to be caused by a germ, but which germ causes it is not yet settled. It differs from rheumatism in that it affects the small joints especially, and leaves some at least of the joints permanently and seriously injured. In some cases there is thinning of the bones and cartilages at the joints (atrophic form), but more often (hypertrophic form) there is considerable increase of both the soft and hard parts of the joints, and especially of the tissues around them; spicules of bone even surround the joint, and the result is limited motion or a complete locking called "ankylosis." Sometimes the whole spine is one rigid bone, a condition called "poker back;" sometimes it is the hips, the shoulders, or the fingers that are "solid." Those pitiful persons who can scarcely move one of their joints are usually afflicted with this disease. It is a disease of adult life especially, but occurs in children. Sometimes it begins insidiously, sometimes as an acute attack which differs little from one of acute rheumatic fever. The patients lose weight and strength. Gradually the joints stiffen, and "crackle" when moved. The joints gradually increase in size, partly because the muscles around them atrophy, but often because they really do swell from the growth of new soft and bony tissue. The

pain is sometimes very severe; sometimes there is none at all. As the joint stiffens it may take some unfortunate attitude,—that is, “contractures” occur. The wrists, knees, ankles, or the whole of the spine, the hips, the shoulder girdle, are the joints most often affected. After one or a few joints become stiff and helpless, the disease ceases, and then the patient, except for the stiffness of the joints, enjoys fine health.

There is no treatment which stops the disease. To patients afflicted with it should be given a full diet, and they should never be tormented with any anti-rheumatism or anti-gout diets. They should live in the fresh air and sunlight. Hydrotherapy, especially warm baths, or a stay at “Hot Springs,” may help. Massage, passive movement exercises, and other means to prevent ankylosis in unfortunate positions, are the important things.

MUSCULAR RHEUMATISM.—This term means much more to the lay mind than to the doctor, for it is a term conveniently applied to any painful conditions of the muscles. Just what the trouble is no one knows. Certain forms are definite enough, as “lumbago,” “stiff neck,” pleurodynia (pain which is felt in the muscles of the chest on each respiration, and which is often mistaken for intercostal neuralgia or pleurisy), and several other varieties which differ only in the location of the pain. These “muscular aches and pains” differ greatly in severity. Their attacks follow exposure to cold—a “draught” for instance—or wet, or hard muscular exertion.

In treatment rest is the important thing. Hot compresses and the Paquelin cautery afford great relief.

CHAPTER XIV

ACUTE DISEASES OF UNKNOWN ORIGIN

UNDER this heading will be considered that group of very contagious diseases which include the so-called diseases of childhood. Strangely enough, the germs causing some of these diseases have not yet been discovered, and those thought to be the cause of others have not yet been sufficiently studied. The evidence at our command now indicates that they are caused by animal micro-organisms rather than by bacteria.

Five diseases—scarlet fever, measles, German measles, smallpox, and chicken-pox—are grouped together as the “acute exanthemata,” or acute fevers with skin rash as an important symptom.

Scarlet Fever.—This very contagious disease is characterized by acute fever, a scarlet skin rash, and a sore throat.

The fever begins in from one to seven (usually in three or four) days after exposure. The onset is very sudden, accompanied in most cases by vomiting, and often, in children, by a convulsion. The temperature rises on the first day to 104° or 105° F. The face is flushed, the skin dry and hot, the tongue furred, and the throat sore. On the second day, sometimes on the first, the skin rash appears. This consists of tiny red dots on a flushed surface and gives the skin a vivid scarlet color. It appears first on the neck and chest, then spreads rapidly and covers the whole trunk in about twenty-four hours. It affects the face least and sometimes not at all. This rash is not a “breaking out,” but an intense congestion, or “erythema,” of the skin. This congestion disappears on pressure, and is gone after death, as the “blood leaves the skin.” The skin is swollen and tense, and often itches intensely.

The tongue is coated along its centre and vividly red on the edges, but its distinctive characteristic is the swollen papillæ, which give it the name “strawberry” or “raspberry” tongue. This is often enough for diagnosis.

The throat is always sore. Sometimes it is only red and swollen, but in severe cases it is covered with a membrane which strongly resembles that of severe diphtheria.

The other symptoms are those of any fever.

In two or three days the rash begins to fade, and it is gone in about a week. The fever diminishes with the rash. As soon as the rash has disappeared the skin, which is dry and rough, begins to peel. This desquamation continues till the whole cuticle is shed, and takes from eight to about fifty days. The skin peels off in fine scales and in large sheets. The process seems to begin on the neck and chest, but the very first traces of it are seen under the finger nails. This may be even a very early symptom of the disease.

No acute infectious fever varies so markedly in severity. It occurs in epidemics,—some of the mildest character, some of the severest. Some patients have no rash at all; others have a rash so slight that it is easily overlooked. Some cases are recognized only after nephritis or some other sequelæ have developed. The most malignant or fulminant cases may die in from twenty-four to thirty-six hours after the first symptom. These cases have a temperature as high as 108° or 109° F., convulsions, and coma. The hemorrhagic cases die in two or three days after the onset, and are marked by extensive hemorrhages, which may cover almost the whole skin. They bleed from the nose, also the kidneys, etc. In other cases the throat symptoms are so severe that the diagnosis of “malignant diphtheria” is made.

Few diseases have more complications and sequelæ than this, and one reason is that *Streptococcus pyogenes* finds an especially favorable environment in scarlet fever patients. Many, indeed, believe it to be the cause, but the fever is more likely due to a tiny animal, traces of which have been found in the skin. Nephritis is the most common and serious complication. It develops in from 10% to 20% of all cases and is the starting-point for very many of those cases of Bright's disease that occur among adults. It develops during convalescence, sometimes as late as the fourth week after the fever is over. It occurs in all grades of severity. The severe arthritis that is often a complication of scarlet fever is usually a streptococcus infection of the joints. The same is true of the complications of acute endocarditis and pericarditis.

The inflammation of the throat often extends to the middle ears, and the otitis media resulting is one of the most common causes of deafness. There is a long list of other complications, but these are the most common.

Scarlet fever is seldom mistaken for any other malady, and other diseases are rarely taken for scarlet fever. Measles is sometimes confused with it, but a child with measles usually feels miserable for several days before the rash appears. The rash of scarlet fever begins, and is especially marked, on the chest, and is different in character from the rash of measles. Diphtheria is a much more difficult disease to exclude, and this cannot be done with certainty unless the diphtheria bacillus itself is found. In scarlet fever the throat may look very much like that in diphtheria; in diphtheria there may be a scarlet hue to the skin; and the two diseases may coexist. After the use of belladonna, quinine, potassium iodide, or diphtheria antitoxin, there is sometimes a rash closely resembling that of scarlet fever. In septicæmia there may be a similar rash.

Concerning the scarlet fever following surgical operations there is much doubt. Its course is like that of a mild scarlet fever, and some think it identical with that disease, others do not.

Acute exfoliating dermatitis is a rare and very interesting disease, which may explain the "second attacks of scarlet fever." It is a fever of sudden onset, with a course of five or six days, a scarlatinal rash, and marked desquamation, but without throat or tongue features.

Scarlet fever occurs in epidemics and chiefly in the autumn and winter,—that is, during the school months. It attacks all ages, but is an especially serious disease for children, 90% of those who die of it being less than ten years old, and by far the most of these under six. It is not really so contagious as measles. It seems to be communicated more by the secretions (of the nose, ear, and throat) than by the scales of the skin. Some blame milk for its spread. A third person seldom carries it. Every patient should be completely isolated for at least two (better three) months after the temperature is normal. The mortality varies greatly, depending both on the community and on the epidemic; it varies from 5% to 30%, but a fair average is 10%.

The treatment is simple, consisting chiefly of good nursing. The hospital is the proper place for the child with scarlet fever, and it were better did law compel him to go there, since so many would thus be saved from having this disease. The child should be nursed in a large, cool room. He should wear a flannel gown. The best diet consists of liquids, chiefly milk and large quantities of water. The bowels should be freely moved, and cold sponge baths should be given when the patient is delirious or the fever high. To prevent complications the child should be kept in bed for at least ten days, and better still a month, after the temperature is normal, to prevent Bright's disease. The throat may need swabbing, the ear-drums puncturing, and the urine should be carefully examined for signs of nephritis. When desquamation begins, the patient should be rubbed almost daily with some oily substance, to prevent the fine scales from diffusing in the air.

Measles.—Measles is an acute fever, which manifests itself chiefly in the skin and upper respiratory tract. Its germ is not yet known.

Measles is an exceedingly contagious disease, and very few escape it. The first symptoms appear in from seven to eighteen days, usually about fourteen days, after the child has been exposed. It begins as a cold in the head, with some fever and malaise, which last from three to six days. The patient feels wretched. Diagnosis is almost impossible during this stage, and yet it is then that the disease is very contagious. Very soon begin headache, nausea (perhaps vomiting), and chilly feelings. There is now considerable coryza, indicated by coughing, sneezing, and redness of the eyes and lids. As the temperature rises, the skin, of the face especially, is flushed and feels hot and tingling. The tongue is furred, the mucous membrane of the mouth and throat is very red, and on the inside of the cheeks may be seen little blue dots, "Koplik's spots," an early and very sure sign of the disease.

On about the fourth day the skin rash appears, first on the forehead, then on the face, then over the whole body. At first are seen little red spots, "like flea bites," which, increasing in number, are arranged in groups, sometimes of crescentic shape. These dots are little papules, very slightly raised above the skin, and can just be felt.

On the fifth or sixth day of the fever the symptoms begin

to abate, and a fine, branny desquamation of the skin begins, which is complete in some cases in a few days, in others in several weeks.

In very severe cases, usually fatal, the rash consists not of little red papules which disappear on pressure, but of minute hemorrhages under the skin (black measles). The patients thus affected may bleed from the mouth also, the bowel, etc.

The complications of measles are very important. Some cases have a chronic coryza with enlarged tonsils and adenoids. We know that tuberculosis is apt to follow measles, and it may well be that such a condition of the throat as measles causes allows the tubercle bacillus to get a foothold. The patients sometimes have severe nosebleed. Some have laryngitis. Ear troubles (otitis media) follow measles more often than they follow scarlet fever. Severe bronchitis and bronchopneumonia are very common and dangerous complications and explain most of the fatal cases of measles. Bad inflammations of the mouth, Bright's disease (often), heart trouble (seldom), severe arthritis, even paralysis, meningitis, and brain abscess may complicate measles.

The diagnosis is very difficult until the rash is out, and yet before that is the time when the disease is especially contagious. The slow onset helps exclude scarlet fever.

The mortality from measles varies from 2% to 10% in most countries, but when introduced into a population which previously has never known it,—that is, “into a virgin soil,”—the disease is terribly fatal. In the valley of the Amazon it is feared more than smallpox.

A case of measles is contagious during its whole course. The disease is spread by the desquamating skin and by all the secretions, including those of the mouth, throat, and nose. The poison clings to the clothes, linen, toys, furniture, etc., which have been in contact with the patient. It is easily carried by a third person. The disease will cling to the patient's room, unless this is properly fumigated, for a long time. It is a hard disease to get rid of if once it breaks out in a hospital.

The patient should be kept quietly in bed. The air in his room should be fresh and cool, but great care should be taken to keep him from catching cold, for bronchopneumonia

is to be feared as a complication of measles, and tuberculosis as a sequela. Hot drinks will help bring out the rash. If the fever is high, cold sponge baths are indicated. Simple cough syrups or paregoric will quiet the cough. During desquamation warm baths and oil rubs are useful.

German Measles.—This acute fever is best described as “having the rash of measles and the throat of scarlet fever,” but it really has no relation to either disease. It is very contagious, but is usually very mild. The period of incubation is two weeks or longer. At the onset there are slight fever, headache, pain in the back and limbs, and coryza.

The skin rash appears on the first or second day, at first on the face, and in twenty-four hours has spread over the whole body. It consists of little pink raised spots arranged in patches. After two or three days it fades.

This fever differs from measles in that there are fewer prodromal symptoms, there is little or no fever, the rash is more diffuse, it is a little brighter in color and is in patches which are less crescentic in shape.

Smallpox.—Smallpox, or “*variola vera*,” was once one of the most dreaded of diseases, but it is now scarcely ever seen and much too little feared. It is an acute fever with a rash which cannot be mistaken.

The first symptoms appear in from eight to twenty (usually twelve) days after the patient has contracted the disease. The onset is sudden, often accompanied by a chill, or, in children, by a convulsion. Then appear three symptoms which, when they occur together, should always lead one to suspect smallpox,—intense headache, intense pain in the back and limbs, and vomiting. The temperature quickly rises to 104° F. or more, the pulse is rapid, and a restless delirium is very common. Next a transitory rash, similar to that of scarlet fever or of measles, may appear.

On the fourth day after the onset the true smallpox rash appears. One sees small bright red spots (“*macules*”) on the forehead and wrists, and in a few hours on the face, limbs, and trunk. These macules soon become “*papules*,”—that is, they become raised and feel like shot in the skin. As these appear the temperature falls and the patient feels better. On the fifth and sixth day of the disease—that is, the second and third of the rash—these papules change to “*vesicles*,”

by the development on each of a cap of clear fluid. This cap is not dome-shaped, but depressed in the centre, or "umbilicated." The fluid in the cap soon turns yellow, since the serum becomes pus; and the vesicles are now called "pustules." They are surrounded by a narrow zone of inflamed skin. This transformation to pustules begins on the face, and is complete over the body by the eighth day of the illness. The "secondary fever" of the disease, the "fever of suppuration," accompanies the pustule formation. The temperature, which was quite or almost normal, rises again, and the general symptoms return. This stage may in a mild case last only about twenty-four hours, but it usually is longer. By the tenth or eleventh day of the disease the fever is gone and convalescence begins. The pustules dry down to "crusts," and these gradually drop off, beginning on the face on the fourteenth or fifteenth day of the disease. These crusts may or may not leave a scar, or "pit," behind them.

Such is smallpox, a disease with initial fever of four days, a pause, and then the secondary fever; and with a skin eruption which passes successively through practically all the stages and forms of skin lesions,—macule, papule, vesicle, pustule, scab, and scar. The rash is *always* most abundant on the face, hands, and feet; somewhat less abundant on arms and legs, and scanty over the trunk in even very severe cases. It is present in the mouth and throat. A point of great importance in diagnosis is that these little skin lesions are all of practically the same age,—that is, one finds only papules or only pustules, etc. This skin rash has a peculiar odor which is unmistakable. One not only can detect it in the sick-room, but can pick out a patient in a crowd. The odor has been perceived in a closed carriage which on the same day had contained a patient.

We have described above a case of "discrete" smallpox. In this the pustules are scattered. In the "confluent" type, however, there are so many pustules on face and hands that they coalesce and form large superficial abscesses. The whole face and head may be frightfully swollen, the features not recognizable, and the skin one huge superficial abscess. This spectacle is one of the most horrible and terrifying ever seen. In such condition are the patients that later are badly pitted.

Confluent cases are much severer than the discrete. The rash of a confluent case often appears a little before the fourth day, and its fever of suppuration lasts much longer than that of a discrete case. There is delirium. In fatal cases death occurs on about the tenth or eleventh day. If the patient recovers, the crusts may cling for even a month.

The "hemorrhagic" smallpox is a very virulent type. In some cases there are only little hemorrhages into the vesicles, but the true hemorrhagic form, "black smallpox," is from the first exceedingly severe. On the second or third day appears an extensive rash of hemorrhages under the skin and even into the eyes. The patient bleeds from the mouth, nose, lungs, rectum, kidneys, etc. He is a frightful object, with skin of a deep purple color, the eyes bloody, the face swollen, etc. These cases die early, even on the third day, before any papules have appeared.

Smallpox was formerly one of the most terrible of epidemics. Practically no one exposed escaped, except those who had survived a previous attack. No age was immune, and the young especially suffered, over 80% of the patients that died being under ten years old. The mortality in epidemics varied much, but for the most it was from 25% to 35%. Vaccination has robbed this disease of almost all its terrors, and epidemics can occur only in an unvaccinated community. Mild cases are common, sometimes among the unvaccinated, but especially in those once vaccinated. It is not uncommon to find among our out-patients cases of the discrete type at the height of the disease, who had a headache a few days ago, but now feel fairly comfortable or come with some minor complaint. These cases are often considered chicken-pox, Cuban itch, etc. Because they are so mild, they are the dangerous cases, for it is they who spread the disease, and those to whom they give it may have the most virulent form.

The cause of smallpox is not surely known, but evidence points to a tiny animal in the skin as the true germ. It is the dust of the skin that seems especially dangerous. The poison clings with great tenacity to clothes, furniture, buildings, etc.

Every nurse should be able to recognize a marked case of smallpox. Any patient acutely ill for less than four days, with severe headache, backache, and vomiting, should be

an object of suspicion, and doubly so if he has no recent vaccination scar. Any person with even a few papules or vesicles or pustules or crusts on forehead and wrists is suspicious, provided these have all recently developed at about the same time and are now all in about the same stage. It makes no difference how comfortable the patient may then feel. Chicken-pox of adults does occur, but so rarely that every case should be detained until seen by a physician and pronounced "not smallpox" before he is allowed to go.

The treatment is chiefly a matter of nursing. The patient must be removed to a suitable hospital. One prescribes the usual treatment for fevers: hydrotherapy, even a continuous bath in confluent cases; liquid diet; morphia for the pain in the back, etc. During the vesicular and pustular stages the affected parts are kept covered with lint masks soaked in ice-water containing some mild antiseptic (dilute mercuric bichloride, for instance), and protected with oil-cloth. Later the crusts are kept moist with vaseline. There is no way to prevent pitting, but the pitting is worse if the pustules and scabs are scratched or picked. Paregoric will check the diarrhoea. The eyes must be kept washed out with boric solution, else the patient may lose his sight. He should be isolated until all scabs are gone.

VACCINATION.—One of the greatest triumphs of medical science is the discovery that vaccination is a protection against smallpox. It has transformed a wide-spread virulent epidemic disease with a mortality of about 35% to a rare disease with a mortality of 6% or 8%. An old and common custom was to inoculate healthy persons from a smallpox case. This, as a rule, produced a mild case of smallpox, seldom fatal, but just as contagious as the worst. Since Jenner's day healthy persons have been vaccinated with lymph from a calf with "cow-pox" ("animal lymph"), or from the vaccination pustule of another person ("humanized lymph"). The disease produced by vaccination, "vaccinia," is not contagious and is scarcely ever serious. Cow-pox is a fever of the cow, with pustular eruptions on the udder. The contents of these pustules are used in vaccinating calves, and from their eruptions is obtained the lymph which is used for man.

Before vaccinating a person the arm or leg is scrubbed

thoroughly with soap and water. The soap is then rinsed off with sterile water. Some prefer to clean the skin with antiseptics, as if for a surgical operation, but this seems to lessen the chance of a "take." The superficial layers of epidermis are scratched a little with a needle, a knife, or an ivory point, or the upper layers of skin are cut with a knife. One wishes to expose the deeper epithelial cells without scratching or cutting deeply enough to draw blood. The lymph is rubbed in with the instrument used in scratching, and allowed to dry, and the place is then protected by a dry sterilized gauze sponge held on with adhesive.

For two days nothing appears. On the third is seen a papule surrounded by a red zone. By the fifth or sixth this has become an umbilicated vesicle, which increases in size until the eighth day. The clear contents of the vesicle change to pus, and on the tenth day we have a large pustule surrounded by an areola of swollen, tender, red skin. On the eleventh and twelfth days the areola disappears; the pus dries; and by the fourteenth the pustule has become a dry brown scab. During the next week this gradually separates and then falls off, leaving a scar. This scar is quite superficial and has a base covered with small pin-point-like holes. The arm and the glands in the armpit are often very sore.

This attack of vaccinia will protect the patient for ten or fifteen years, at the end of which he should again be vaccinated.

If the vaccination follows any other than the above described course, the result must be considered doubtful. If one is not careful, he may get an infection with pus organisms. The arm is then very sore, and the armpit especially so; the scar will be deeper and more conspicuous than desirable, with a shiny base and thick margin.

As for the inoculation, by vaccination, of real disease, as tuberculosis, lues, lockjaw, etc., while this may have occurred, it is very rare and might have been avoided by ordinary cleanliness and the use of good lymph.

Infants are vaccinated after the second or third month. Any one exposed to smallpox should at once be revaccinated. Vaccinated persons, especially those with old scars, may catch smallpox, but it will be the mild "varioid," as a rule.

Chicken-Pox.—Chicken-pox, or "varicella," is an acute

contagious disease characterized by an eruption of vesicles. The period of incubation is from ten to fifteen days. The onset is often with a chill, vomiting, and pain in the back. Children, as a rule, suffer little, but in adults the constitutional symptoms are sometimes severer than in mild smallpox. Within the first twenty-four hours of fever the rash appears. First are seen red papules, then vesicles full of clear fluid, superficial and usually without an areola of inflamed skin. In two days the fluid has turned to pus,—that is, the vesicles are become pustules. Two days later the pustules dry down to dark-brown crusts, which fall off, leaving as a rule no scar; but if the patient scratches them, scarring follows. Since successive crops appear at intervals of from one to four days, all stages of the rash are present at the same time.

This eruption appears first on the trunk, back, and chest; it seldom begins on the face. The pustules never coalesce. They vary in number from a dozen or more to several hundreds. Some of them form in the mouth. Complications are rare, but gangrene of, or hemorrhages into, the pustules may occur. Nephritis and paralysis have been reported.

The diagnosis is easy and can be made early, as the vesicles of chicken-pox are unlike those of smallpox. The former are little translucent blisters, very superficial on the skin, and without areola. They appear first on the trunk. In smallpox the vesicle is the cap of an inflamed papule, and the rash appears first on the face. All stages of pustule formation are found at the same time, which is not true of smallpox, and the rash appears earlier than in the latter disease. But after the first few days it is often very difficult to make a correct diagnosis, since mild smallpox is very like chicken-pox. Several recent severe epidemics were due to the mistake of diagnosing cases of mild variola as varicella.

The treatment is simple because it lies chiefly in protecting others, but difficult because the child strenuously objects to the necessary six weeks' isolation. Chicken-pox is an extremely contagious disease, especially among children. Very few cases have proved fatal, and there is doubt as to the correctness of the diagnosis of these cases.

Mumps.—This very common contagious disease is an inflammation of the salivary glands, especially the parotids, whence the scientific name "epidemic parotitis," and is caused by some germ not yet discovered.

The attack begins two or three weeks after the child has been exposed, with fever, and swelling of one parotid gland. The position of this swelling is very important in the diagnosis; it is just below and in front of the ear, and lifts it a little. Swollen lymph-glands are situated a little lower in the neck. Sometimes the swelling of the parotid is preceded by a few days of fever and malaise, and "grippe" is suspected. Sometimes the first sign is a sharp pain experienced on swallowing anything sour. The swelling increases for about two days, during which usually the other parotid and the submaxillary and sublingual glands also become swollen. The mouth can scarcely be opened, and there is pain on swallowing. After seven or more days the fever and the swelling gradually subside. Some children are scarcely ill at all; some patients, especially adults, are very ill.

The patients, especially those beyond childhood, should be kept quiet in bed, not only until the temperature is normal, but until the swelling is entirely gone. This, though difficult, is the only safe course, since serious complications—orchitis, etc.—may follow.

The disease is communicated directly, probably not by a third person, but patients have infected others even six weeks after the fever is gone.

The treatment is rest in bed, and liquid diet. Hot or ice compresses are applied to the swellings. Cold sponge baths are very agreeable while the fever is high.

There are several OTHER FORMS OF PAROTITIS, which are not contagious, such as that due to lues, or the infectious diseases, that of typhoid fever, of pneumonia, etc., and that following operation. In these cases the gland often suppurates and must be opened.

Whooping-Cough.—Whooping-cough is an acute contagious bronchitis, the germ of which is not yet fully identified. It occurs in all seasons, but sometimes in definite epidemics in the winter and early spring.

The first stage, or "catarrhal stage," of the disease begins from seven to ten days after the disease is contracted, like an ordinary cold in the head, with slight fever, running eyes, and a cough of increasing severity. As a rule, no suspicion of the real diagnosis is aroused during this week. Then begins the "paroxysmal stage," which dates from the first "whoop."

The paroxysms of coughing during this stage cannot be mistaken. A cough is a violent expiration designed to blow something out of the bronchi, trachea, or throat. In the ordinary paroxysms of coughing we inhale after each two or three coughs, but in the paroxysms of this disease the child attempts the impossible feat of coughing fifteen or twenty times during one expiration. Of course the lungs and all the other organs in the chest become forcibly compressed by the strong muscles of expiration. The child then "recovers its wind" in one long inspiration, which it draws with a "whoop." The result of this paroxysm may be the expectoration of a little mass of very sticky mucus. During these paroxysms the face becomes blue, the veins swell, the eyes bulge out, their whites are injected, and it looks as if the child must suffocate. Some children vomit at the end of a paroxysm, and so often during the day that they almost starve. A child may have very few such paroxysms or even a hundred a day. The paroxysms are induced by swallowing, by any irritation in the throat, and by the emotions. The paroxysmal stage lasts from three to four weeks; then the whooping becomes less frequent and finally stops.

The complications of this disease are many. During the paroxysms the strain on the lungs, heart, and blood-vessels is terrible. A blood-vessel may burst, and the child bleed from his nose, eyes, ears, or lungs; or the bleeding may be under the skin, or into the brain, with paralysis as the result. The lungs are often injured; sometimes they become emphysematous, sometimes they literally burst.

The worst complication is an extension of the inflammation from the finest bronchial tubes to the air-cells, causing bronchopneumonia. It is this complication which makes whooping-cough one of the most fatal of the acute infectious diseases of children under five years old and a very serious disease for the aged.

The treatment is, first of all, to isolate the child perfectly and for a long time. While whooping-cough is probably most contagious during the catarrhal stage,—that is, in the week before which the diagnosis is made,—yet it is also contagious for some weeks after the acute illness is over, and the child should not be allowed to associate with other children until some weeks after he is apparently well.

While the paroxysms are severe the child should be kept in bed. During the whole course he should be in the open air. In some hospitals the beds of patients with whooping-cough are outdoors all day, in others they are on the porch day and night.

Of drugs, paregoric should be freely given. Broncho-pneumonia, when it is a complication of whooping-cough, usually develops just at the beginning of convalescence, and it is at just this period that greatest care is necessary.

Convalescence is tedious, but watchful care should be taken that the child does not catch cold or overdo. The chronic cough which sometimes follows is best treated with fresh air, large quantities of good food, and, if possible, a change of climate.

Yellow Fever.—The yellow fever is a disease whose most important features are as follows: a very early jaundice (hence the name) and early albuminuria; a slowing pulse with a rising temperature; and the tendency to hemorrhages, especially from the stomach. The toxæmia varies greatly in different cases.

This "tropical fever" has been endemic for generations in the West Indies and in Central and South America, and is justly called an "American disease." There is good reason to believe that it is always smouldering among the young negroes of these countries, who have inherited a relative immunity, and in whom the disease is so mild that they are scarcely ill. But when the disease breaks out in a non-immune population, it works terrible havoc. It has swept as an epidemic repeatedly over our Southern States to as far north even as Philadelphia. Epidemics of it occur in the warm, wet months, limit themselves to the seacoast, and are especially severe in dirty, crowded districts of large cities.

The germ has not yet been discovered, but the mosquito transmitting this germ has, beyond question, been identified. This mosquito, *Stegomyia fasciata*, is distinguished, among other ways, by his striped yellow-and-black legs. After one of these mosquitoes bites a patient with yellow fever, the germ must develop in its body during at least twelve days, during which it cannot transmit the disease to another person. After that time its bite is dangerous. The disease is not spread by the air, infected clothes, etc.

The period of incubation lasts usually for three or four days after the mosquito's bite. The onset is sudden, as a rule, usually in the early morning, and is accompanied by chilly feelings, headache, and pain in the back and limbs. At the very onset the face is flushed and the slight jaundice has already appeared. The temperature rises rapidly, remains elevated from one to three days, then falls by a lysis which lasts two or three days (the "stage of calm"), then rises again for the "secondary fever," which lasts from one to three days. In fatal cases, however, the temperature remains elevated. While very early in the attack the temperature is rising, the pulse becomes slower, and at the height of the disease may be from 70 to 80 to the minute, and during deferescence as low as from 30 to 40.

Albuminuria may appear, even in the mildest cases, as early as the third day. In severe cases nephritis may dominate the clinical picture.

Vomiting is common from the very first, and later is characterized by much blood ("black vomit"). Hemorrhages from various other organs are very common.

The mortality of various epidemics has varied from 15% to 85%. When severe yellow fever is one of the most fatal of epidemic diseases. In some epidemics the disease is very mild.

The prophylaxis is a matter of prime importance to the nurse, for the control, or lack of control, of the epidemic depends on her—not on the doctor. Suppose a case of yellow fever is in the bed before us. That man *per se* is not at all dangerous, nor will the many *Stegomyias* which are humming about the room be dangerous until twelve days have passed. The diagnosis once made, the problem is to prevent, by means of screens, all mosquitoes from biting our patient and to prevent those which have already bitten him from escaping from the house. During the next twelve days every mosquito in the house should be killed. If this is done, no one can get yellow fever from our patient.

The patient is made comfortable by cold baths, but great care must be taken that he does not catch cold. The question of diet is difficult because vomiting is often so severe; nutritive enemata are often necessary. Various remedies, including cracked ice, may control the vomiting, while stimulants are often necessitated by the very collapsed condition.

Dengue.—This pandemic fever of tropical and subtropical regions, including our Southern States, is interesting because "there is no disease, not even influenza, which attacks so large a proportion of the population" (Osler), and also because it sometimes so resembles yellow fever that there is still doubt as to whether certain recent epidemics were dengue or yellow fever.

The disease may be briefly described as a febrile paroxysm characterized by severe pain in the joints and muscles and by a skin rash. The cause is not known. From its duration some call it "three-day fever." The pains in the head, eyeballs, back, limbs, and in one, many, or all of the joints are so exquisite that they have given the disease the name of "break-bone fever." It is followed by stiffness of the muscles and joints, which so changes the facial expression and the gait that the disease is also called "pantomime fever," and "dandy fever." There is often a skin rash of very variable character. This disease is seldom fatal.

Typhus Fever.—Typhus fever, called also "hospital fever," "camp fever," "spotted fever," "jail fever," and "ship fever," was formerly "one of the great epidemics of the world." It occurred wherever starving human beings were overcrowded amid filthy surroundings, and wherever there were war, famine, and misery. While 95% of the victims were of this class, and their mortality was from 12% to 20%, the fever seems to have been especially fatal to the doctors and nurses attending these patients. It was present in every large city until about fifty years ago, but since then has been a curiosity. Its germ has not been discovered, but it has disappeared before the better hygiene. For a long time no distinction was made between it and typhoid fever.

Briefly described, it is a very acute and extremely contagious fever, with an incubation period of twelve days, and a sudden onset with chill, pain in the back and legs, and unusual prostration. On the third, fourth, or fifth day appears the macular rash, on the abdomen first, but in two or three days over the entire body. Even in the mildest cases the macules are, many of them, replaced by tiny hemorrhages into the skin, or "petechiæ," which give the disease the name of "spotted fever." The temperature is very high, even 107° F., and very constant. There are always profound

nervous symptoms; prostration, delirium or even mania, and, in fatal cases, coma. In cases which recover there is crisis at the end of the second week, with rapid convalescence. The treatment is that of typhoid fever.

Beriberi.—This disease is really a multiple neuritis, or inflammation of the nerves. It is very common in the far East, often occurring in epidemic form, but has been found in almost every country, including this. There have been cases in Massachusetts, Alabama, and Arkansas. The cause is not known, but poor food is a very important factor.

The symptoms are pain and weakness in the limbs, and even paralysis, together with tingling and loss of sensation in these parts. In some cases there is wasting of the muscles of the limbs—the “dry form;” in others there is first a dropsy of the limbs or of the whole body, with fluid in the abdomen—the “wet form,” and then, when the dropsy disappears, the wasting is apparent.

In the Far East the mortality varies from 2% to 50%.

Hydrophobia.—This condition, called also “rabies,” is an acute, fatal, infectious disease, which may attack almost any animal, and man. We are sure it is caused by a specific germ, which localizes in the brain especially and which travels along the nerves. The disease is communicated from animal to animal or man through the saliva. Dogs are very susceptible to it, but the bite of a mad wolf or cat is more dangerous than that of a mad dog. The disease among men could be entirely eradicated by rigidly enforced laws to muzzle all dogs. Only about 15% of persons bitten by mad dogs contract the disease. The danger depends greatly on the depth of the bite, and whether the animal bites through the clothes or not; the worst bites are those on the face or hands.

In man the period of incubation of the disease is from two to eight weeks long, seven weeks being the average length. It is during this period of incubation that we have a chance to prevent the development of hydrophobia.

After the period of incubation the disease develops in three stages. First is the “premonitory stage,” during which the flesh around the bite becomes inflamed and the patient mentally depressed and very irritable. Then comes the “stage of excitement,” in which even the slightest sensations produce violent spasms, especially of the throat and mouth.

The attempt to swallow water produces spasms of the throat, and hence the patient hates the sight of that liquid. He sometimes has maniacal attacks, during which he is dangerous; but in man this is rare. After from one to three days of this stage begins the "paralytic stage," which lasts but a few hours—from six to eighteen. The patient gradually becomes comatose and dies.

When a person is bitten by a suspected dog, the animal should be at once killed, and its brain and spinal cord sent to the proper authorities, who will inject a little of it into a rabbit. If the dog was rabid, the rabbit will become so in from fifteen to twenty days. Treatment of the bitten person, however, should begin as soon as possible after the injury.

Pasteur found that the virus of hydrophobia can be made so virulent that if inoculated into a rabbit it will, instead of taking fifteen or more days to make the animal mad, do so in seven. The dried spinal cords of rabbits that have died from this very virulent form are the remedies used in treating persons. A little of one of these cords, which has been dried to weaken it somewhat, is injected into the patient; the amount used is not enough to make him very ill. A certain amount of immunity is thus acquired. Then a little larger dose is given, then a larger still, and so on, the immunity being thus increased until he can stand without any injury a dose of intense virus which had it been given on the first day would surely have caused a rapidly fatal hydrophobia. Meanwhile the virus which the dog's bite injected into him, and which takes more often seven weeks to develop, is become ready to produce hydrophobia. But the man is already immune to a more virulent form of the disease than the dog's bite could give, and so the bite does no harm. The results of the Pasteur treatment when well given are very satisfactory.

If this treatment is impossible, the bite should be cauterized with carbolic acid or caustic soda, and the wound kept open. It should be remembered that in only one person out of six that are bitten is the disease likely to develop. If the disease does develop, the outlook is hopeless. The patient is then kept as quiet as possible in a dark room, and morphia, chloroform, and other strong sedatives are used without stint. If the spasms of the throat cannot be relieved by cocaine, the patient should be fed per rectum.

CHAPTER XV

ANIMAL PARASITES

Most of the diseases already studied are caused by minute plants. Other diseases are caused by equally primitive and simple parasites,—the minutest animals, or “protozoa.” It is easy to study the bacteria and to grow them in glass tubes, but the tiny animals cannot be seen, studied, and grown in

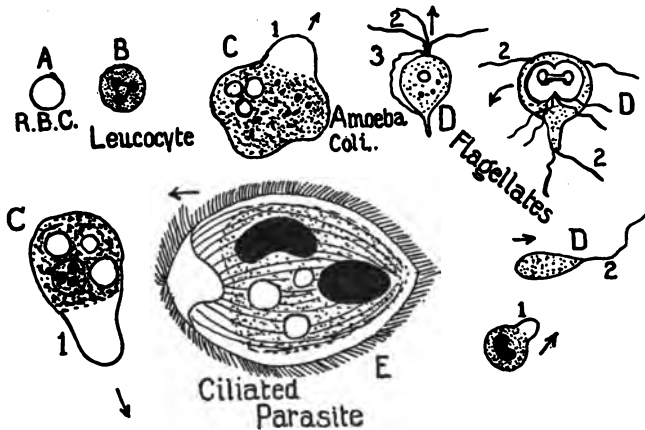


FIG. 111. Some of the smaller animal parasites. A, red blood-corpuscle, and B, leucocyte, introduced for comparison of size. C, amoeba coli, 1, pseudopod. The arrows in all cases show the direction of movement of the organism. D, flagellated protozoa, 2, flagella 3, membrane. E, ciliated protozoan. In the lower right-hand corner is a leucocyte in motion; note the contrast in size and appearance between this and C. In the lower left-hand corner is an amoeba coli which has devoured red blood corpuscles and a leucocyte.

the same way, and we do not yet know well how to handle them. There are a few diseases which we now know are caused by these minute animals. Their number is increasing, and will doubtless increase still more, as our methods of work become more accurate. Amoebic dysentery, some forms of diarrhoea, malaria, the sleeping illness of Africa, are surely due to protozoa. It seems likely that the germs of smallpox

and scarlet fever have been discovered, and, if so, that they are protozoa. By analogy we may suppose that yellow fever, measles, and the other most contagious diseases are also caused by animals.

Amœba Coli.—Amœbic dysentery is caused by a minute protozoon, *Amœba coli*, which resembles a leucocyte in many ways, except that it is from two to ten times as big (Fig. 111, C). Its nucleus is small and round, and is seldom seen because its protoplasm is full, not of granules, as are the commoner leucocytes, but of fragments of food which the parasite has eaten,—of leucocytes, red cells, bacteria, etc. This amœba moves very rapidly on the glass under the microscope.

If Amœbæ coli gain an entrance to the body, they settle in the large intestine. Here they burrow into the mucous membrane, in which they multiply, and spread their burrow for some little distance under the mucosa. The result is a pocket with numerous branches extending a considerable distance in all directions, with a protecting roof of mucosa, and with only a small orifice opening into the bowel. This roof of mucosa soon sloughs off, or, tearing off at one end, it may hang as a rag, leaving exposed an ulcer, which from its shape and its method of formation is known as an “undermining ulcer.” The depth of these ulcers varies. Usually their floor is the muscle wall of the bowel, but they may perforate the entire wall and cause a fatal peritonitis. The large bowel and the lower part of the small bowel may be covered by these ulcers, and very little normal mucosa be left.

The symptom of such a case is dysentery,—that is, the passage of frequent small stools in which there are much blood and mucus. If a speck of this mucus is examined, it may be found to be simply alive with these amœbæ. There is fever, and often there is rapid emaciation. The important features of these cases are their chronicity and their tendency to recur. Under the treatment dysentery promptly stops, but in a few months it begins all over again. The amœbæ lie well protected in the bowel wall, quite out of reach of medicines or bowel irrigations, and may remain quiet there for years.

The treatment is to wash out the bowel once or twice a day with large quantities (about two quarts each time) of a fluid which will kill amœbæ. Dilute solutions of quinine

(1:1000) or of silver nitrate are the fluids generally used. These readily kill the amœbæ in the exposed ulcers,—the amœbæ which cause the symptoms,—but do not reach the well-protected foci, which are causing no symptoms. The enemata should be given with the patient in the knee-chest position and should be retained as long as possible. The object is to wet as much of the bowel as possible with the fluid.

To diagnose this form of dysentery, one must find the living amœbæ and see them move. The nurse can be of great help to the doctor in this search. A dose of salts is given early in the morning, or on the evening before the examination, to loosen the stool and wash down mucus from the upper colon. The amœbæ very soon cease their movements and are not to be recognized when the stool is cool; everything, therefore, should be ready for the examination before the stool is passed. A vessel or bed-pan warmed to about blood heat (not a hot pan, for that would "cook" the parasites) should be used. A particle of mucus, especially bloody mucus, or the liquid part of the stool, is chosen for examination. A better method is to pass a rectal tube or a rubber catheter as far up into the rectum as possible. A particle of mucus will usually be found in the eye of the tube, which must be kept warm.

The amœbæ in a large number of cases (about one-fifth) make their way into the portal veins and are carried to the liver (see page 140), where they cause abscesses. These abscesses differ from those due to pus-producing bacteria in that the amœbic abscesses are more often single than multiple and are very large. There is little real inflammation about them; they are large holes full of decayed and liquefied liver tissue. In their necrotic walls are hosts of the amœbæ. These are the abscesses whose contents often pour through a perforation into the lung and are expectorated. One point to be emphasized is that these abscesses are frequently found in patients who have had almost no dysentery, and in some who have had none at all. The amœbic infection of the intestine was too slight to produce symptoms, and yet some of the parasites found their way to the liver.

Amœbic dysentery is a disease of the tropics, especially Egypt, India, and the far East, but also is common in Baltimore and even farther north. That the parasite is swallowed

with drinking water there is little doubt. Harmless amœbæ are common enough in stagnant water, but the parasite that causes amœbic dysentery has learned, like pathogenic bacteria, the trick of living at the expense of a host.

Ciliata and Flagellata.—In the stools may often be found other parasites, those belonging to the ciliated and flagellated groups. They seldom, if ever, do any harm, although when

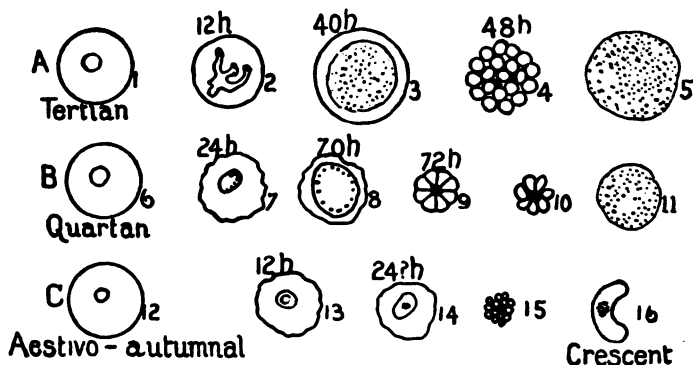


FIG. 112. The malaria parasite. (Magnified 1000 times.) A, the parasite of tertian malaria. 1, a young hyaline which has just entered the red corpuscle. 2, the same 12 hours later. The parasite is larger, actively amoeboid and now contains pigment granules. 3, the same when 40 hours old. The red corpuscle is swollen, the parasite is large and contains much pigment. 4, the same when 48 hours old. The red corpuscle has disappeared. The parasite has divided into twenty small parasites called "segments." These will separate and make their way into other red corpuscles, as was the case with 1, and start the cycle over again. 5, some of the parasites like 3 do not segment but grow to a form which will develop further only in the mosquitoes' stomach. There 5 will grow, divide into thousands of tiny forms, which if injected into a person's blood by the bite of this infected insect will like 1, start the cycle. B, the parasite of quartan malaria. Note the differences in ages, in size, the different effect on the red corpuscles which shrink, and the coarser granules of pigment. 9, the marguerite form, or "pre-segmenter" which divides to only about seven segments. 11, is similar to 5. C, the parasite of aestivo-autumnal malaria. Note the differences in ages, in size, in the effect on the red corpuscles, and the peculiar shape of the parasite, 16, which like 5, and 11, is designed for the mosquitoes' stomach. (The segments of 15 are drawn too small.)

very numerous they do cause a diarrhœa which in some cases has proved fatal (Fig. 111).

Plasmodium Malariae.—Malaria is an acute infectious disease caused by animal micro-organisms of three slightly different varieties, all grouped under the term *Plasmodium malariae*. These malarial parasites are protozoa—that is, they are the simplest animals—and strongly resemble a leucocyte, except that they are much smaller. One of the three varieties is called the tertian parasite, because the cycle of its life takes just forty-eight hours and so ends on

the third day. The quartan is so named because its cycle always takes seventy-two hours, and so is completed on the fourth day. The æstivo-autumnal parasite, so named because the form of malaria produced by it occurs especially in summer and fall, has a cycle which varies between twenty-four and seventy-two hours. In the following pages we shall specify the individual varieties by the terms tertian, quartan, etc., when necessary, but the term "malarial parasite" will always include all three (Fig. 112).

The malarial parasite lives in the red blood-corpuscle. When young it attaches itself to a red cell and makes its way to the cell's interior, where it is well protected from the hostile blood-plasma, as well as from medicine. There it grows, consuming the hæmoglobin as its food. When full grown it fills the cell, which now is merely a hollow shell. The parasite next splits up into from ten to twenty small parasites ("hyalines," "segments"), the shell bursts, and then young parasites scatter in the blood stream. For a short time they lie free in the plasma, where most of them die; a few fortunate ones find their way into new red cells. When they are once inside cells, the process, described above, repeats itself; the young parasite grows to maturity and then segments.

The three varieties of malarial parasites differ somewhat in appearance, size, the effect they have on the cell in which they live, and in the number of "hyalines" into which they split; but we cannot dwell on these differences here.

In the case of the tertian parasite the cycle takes forty-eight hours. In the blood of a patient with "single" tertian malaria there may be hundreds of millions of these parasites at one time, but they are all of the same age. They all get full-grown and divide ("segment") at about the same time, and so for a few hours the blood contains very many adult forms and also young "segments." It is just then that the chill and the sharp rise in temperature occur, and then that quinine will kill the parasites. This patient will have a chill every second day. But often there are two groups of the tertian parasites present at the same time. This is like having two diseases at once, for the two groups grow independently. The members of one group will always be just twice as old as those of the other, and, since each group will cause

a chill every other day, the man will have a chill every day ("quotidian fever").

The cycle of the quartan is seventy-two hours long, and hence, if but one group is in the blood, the man will have a chill every fourth day,—for instance, on Monday, Thursday, Sunday, Wednesday, etc.; if two groups are present the chills might come on Monday, Tuesday, Thursday, Friday, Sunday, Monday, etc.; if three groups, then he would have a quotidian fever.

The æstivo-autumnal parasites do not long remain well grouped. They may be so at first, and then the chills will occur every day; but soon all ages will be present at the same time, there will always be some segmenting, and so the temperature will be irregular or even continuously elevated, and the patient will have only "dumb chills" or none at all.

We have spoken thus far of only those forms of the parasites which cause the fever in man. But other forms also are always present. Not all of the segments repeat the process described above. Some develop into adults which do not segment, but remain, perhaps for months, unchanged in the blood. These are the "sexual forms." They do man no harm. But suppose a mosquito of the *Anopheles* group to bite the patient. The blood it sucks will contain many of the ordinary malarial parasites and a few of these sexual forms. The former will die. The latter will develop further in the mosquito, and in a few days will reach their maturity and split into myriads of young. The next time that mosquito bites, these little ones will be inoculated into the man's blood along with the mosquito's saliva, at once enter red corpuscles, and start the cycle already described; that is, they will grow and divide, and the divisions will enter new cells and will themselves grow and divide, etc., etc.

For several days after the mosquito's sting the man has no symptoms, for at first there are not enough parasites to cause a chill. They continuously increase in number until on some day the man has chilly feelings; perhaps the next time segmentation occurs they will be numerous enough to cause a real chill.

There is only one variety of mosquito which can act as the intermediate host between man and man, and that is the *Anopheles*. This can be recognized if seen when resting

on a wall, for, instead of standing "hunchbacked," as does the ordinary mosquito (*Culex*), it stands with body, thorax, and bill in a straight line forming with the wall an angle of about 30°.

Patients with tertian and quartan malaria (the "regular intermittent fevers") feel perfectly well on the days when they have no chill. The chill begins quite suddenly, with sensations of cold, shivering of the whole body, and sometimes nausea and vomiting. The face and hands are blue, and there is often intense headache. The temperature rises rapidly to 104° or even to 107° F. After about ten minutes the patient warms up, and presently he gets intensely hot. His skin is flushed, his heart beats violently, he has a throbbing headache, and he may even be delirious. This stage lasts from thirty minutes to four hours, and during this time the temperature stays at its highest point. Then the patient begins to perspire profusely, the headache ceases, he feels better, and soon is entirely relieved. The entire paroxysm lasts from ten to fourteen hours. These paroxysms usually occur during the daytime, and, oddly enough, they generally begin in the forenoon. Each of them leaves the patient a little weaker and more anæmic than before.

Æstivo-autumnal fever may at first cause regular chills, but later the temperature curve may be almost as straight as that of typhoid fever. The symptoms are much less marked than in the tertian or quartan fever, and yet this form of malaria is much more serious, for there is far more destruction of blood. The patient becomes slightly jaundiced. He may in very severe cases become comatose and die (pernicious malaria), or develop a severe nephritis or severe gastrointestinal or brain disturbances. In the last two cases the parasites accumulate in the blood-vessels of the organs affected. Patients with this form of malaria may become extremely anæmic and emaciated. Some pass large quantities of hæmoglobin in the urine, the color of which gives this condition the name of "black-water fever."

To avoid contracting malaria one must keep free from the bite of an infected *Anopheles* mosquito. The insects bite usually after sundown. Houses should be well screened, and nowhere should there be allowed to remain little pools of stagnant water where mosquitoes can breed. Patients with

malaria should be screened, in order that the *Anopheles* mosquitoes may not bite them and thus make them hosts of the germ.

The cure for malaria is quinine. The patient should lie in bed as long as there is any fever; for malaria, especially the æstivo-autumnal form, is as serious as is almost any of the acute infectious diseases, as regards both its own immediate danger and the dangers of its sequelæ. The quinine should be given in liquid form, because much of that given in pill form is not even dissolved in the intestine. It should be given in regular doses, whether the patient has a fever or not; and, what is most important, quinine should be taken for at least three months after the last trace of fever is gone, for this parasite may rest hidden in the bone-marrow and spleen for years, ready to cause a recurrence of the disease when the proper conditions are present. These hidden parasites become very active after an accident, for instance, and at certain seasons of the year. Malaria is very common in the spring, and yet the probability is that every spring case is a recurrence of an infection of a year or more before.

In the cases of tertian and quartan fever the infection is best controlled by having considerable quinine in the blood (about 30 grains a day) at the time when a chill is expected. This chill will occur, but the majority of the parasites will be killed by the quinine during the chill while they are free in the plasma, but this will be the last for a while. Stop quinine then, and the few parasites left will increase in number until enough are present to cause a chill. Those hidden away are hardest to kill, whence the need of continuous treatment for months (about five grains a day).

The diagnosis of malaria is simple. The word "malaria" should never be used unless one has found the parasites themselves in the case under consideration. And yet the term is commonly employed for any disease with chills. Cases of typhoid fever, of abscess formation, and especially of very early tuberculosis are long treated for malaria, to the great disadvantage of the patients, for valuable time is thus lost; pounds of quinine are wasted yearly on such cases. It may be said, in general, that a fever which a few consecutive doses of quinine will not stop, temporarily at least, is not malaria. The examination of the blood is important

not only to prevent our mistaking these other conditions for malaria, but also to avoid mistaking malaria for other conditions; for the course of a case of æstivo-autumnal fever is often as unlike a "malaria" as one could imagine. In the pernicious cases the quinine is injected directly into a vein.

Trypanosoma Gambiense.—This terrible disease of the tropics, TRYPANOSOMIASIS, also called "sleeping sickness," is due to an animal parasite recently discovered, the *Trypanosoma gambiense*. This little animal is one of the protozoa and has a long flagellum by means of which it swims actively, somewhat like an eel, among the red blood-corpuscles. It is a little longer than one of the corpuscles is broad. Trypanosomiasis is a terrible scourge for the animals of India and Central Africa. In some parts of these regions cattle and horses cannot live at all. The parasite is introduced into the blood by the bite of a certain fly (Fig. 101, E).

When this parasite infects a man, it multiplies in his blood without causing any symptoms. Later begin fever, loss of weight, and swelling of the lymph-glands. After a few months the parasite invades the brain and spinal cord, and then comes drowsiness, presently coma, and finally death. Arsenic seems to cure some cases.

Leishman-Donovan Bodies.—PYROPLASMOSIS, TROPICAL SPLENOMEGALY, DUM-DUM FEVER.—This disease is also due to a minute animal parasite recently discovered. Its chief symptoms are an irregular fever, large spleen, anæmia, hemorrhages, and œdema. About one-third of the cases die. These cases were always thought to be a form of malaria which did not yield to quinine, until doctors refused to say "malaria" before they saw the malaria parasite. Then they found this parasite. This is a tropical disease, occurring especially in India and Egypt.

Filaria Bancroftii.—FILARIASIS.—The embryos of the common filaria are found in the blood only during the night. They are easily seen with a microscope, as they are relatively large (about $\frac{1}{15}$ of an inch long) and move very actively, driving the red corpuscles in all directions. None are found during the day; they are then all in the capillaries of the lung, liver, and other internal organs. Towards evening a few appear, and then they increase in number until about midnight, when most are present, then decrease till sunrise.

Another variety of this worm has embryos which are free in the blood during the day only, while those of a third variety are present day and night. We do not know how these embryos time themselves so accurately, but their appearance surely has some relation to the man's sleeping hours, for, if a day-laborer becomes a night-worker, these embryos also change their hours.

This parasite is very common in some countries, especially British Guiana, but it is rather common in our Southern States also. We know that it is introduced into the blood by the bite of a mosquito, because the embryos can be found in the proboscides of some mosquitoes. These insects doubtless get infected by sucking the blood of a patient with filariasis. The young embryos which the mosquito injects take up their home in the lymph-channels of the patient's pelvis and there grow to maturity, reaching a length of several inches. They block up the lymph-vessels. The result of this may be that the lymph is dammed back into the skin of the legs, with "elephantiasis" as the result,—that is, the leg becomes huge from the great thickening of the skin. Or the worms block the lymph-channels from the bladder, and blood and lymph rupture into the bladder, and the patient's urine at times is like milk, or full of blood, or contains both blood and lymph. Or tumors resembling hernias form in the groin. The adult worms produce the enormous numbers of young embryos which flood the blood, but which themselves do no harm, for they do not develop further.

The only possible treatment is to remove the adult worms by a surgical operation.

Trichina Spiralis.—TRICHINIASIS.—This parasite is very often found in America, but the disease trichiniasis is much rarer than in Germany, because so few persons here eat raw pork. The muscles of almost 2% of the pigs in this country contain the embryos of the worm. These little embryos lie in the muscles, well coiled up, and surrounded by a fibrous wall. They can be just seen, like tiny grains of sand, in the meat. As long as they remain there the embryos will develop no further and will do the pig no harm. But if pork containing the germs of trichiniasis is eaten raw, or so little cooked that the embryos are not killed, the gastric juice of the person who eats the pork will digest the fibrous wall around the

embryos and set them free. During the next week these embryos grow in the man's intestine to adult worms and make their way into the mucous membrane. Here are born myriads of young, which at once enter the blood stream and are carried in the blood to all parts of the body. They settle in the muscles especially, and there, in about six weeks, become encysted, just as in the pig. No further harm is done by them, although they may be alive at the end of twenty-five years.

It is while all these embryos are being carried by the blood and are wandering about that the symptoms occur. The man has a fever suggesting typhoid, pain and soreness in all his muscles, and there is œdema of the eyelids. Many of the patients die, some are invalids for years, some show scarcely any symptoms at all.

The diagnosis is suggested by the patient's admission that he has been eating raw pork and by the symptoms; but, in order to be sure, one must remove a little piece of muscle and find the embryos themselves.

The treatment, possible only when the diagnosis is made very early, is to purge the patient, in order to get rid of the worms still in the intestines. After that there is nothing more to do.

Ankylostoma Duodenale; Uncinaria Americana ("Hook-worm").—ANKYLOSTOMIASIS OR UNCINARIASIS.—This disease is caused by two quite similar little intestinal worms (Fig. 113, C) about half an inch long, *Uncinaria americana* and *Ankylostoma duodenale*, which live in great numbers in the upper part of the small intestine. Their chief occupation seems to be to bite the mucous membrane in many places and thus cause a great deal of bleeding. It is doubtful whether or not they use this blood for food. The result of all these minute hemorrhages is a severe secondary anæmia, which bears various names,—hookworm disease, miners' anæmia, Egyptian chlorosis, tunnel-diggers' anæmia, brick-makers' anæmia, etc. It is one of the most fatal of parasitic diseases. It occurs very commonly in our Southern States, where it is called "Southern anæmia," "anæmia of the poor whites," "sleeping sickness," etc. One jocular reporter named the worm the "germ of laziness." The embryos of this worm live in the dirt, sand, and clay, and easily infect a man both by mouth, if he eats with dirty hands, and by boring through the skin,

causing, while doing so, "ground itch." The patient is anæmic, and, if a child, develops to maturity slowly; the skin is of a muddy, pale hue ("Florida complexion"); the eyes are dull and heavy ("fish eyes"). Later the skin is cedematous, and the patient very weak and short-breathed. Many patients that are not treated die.

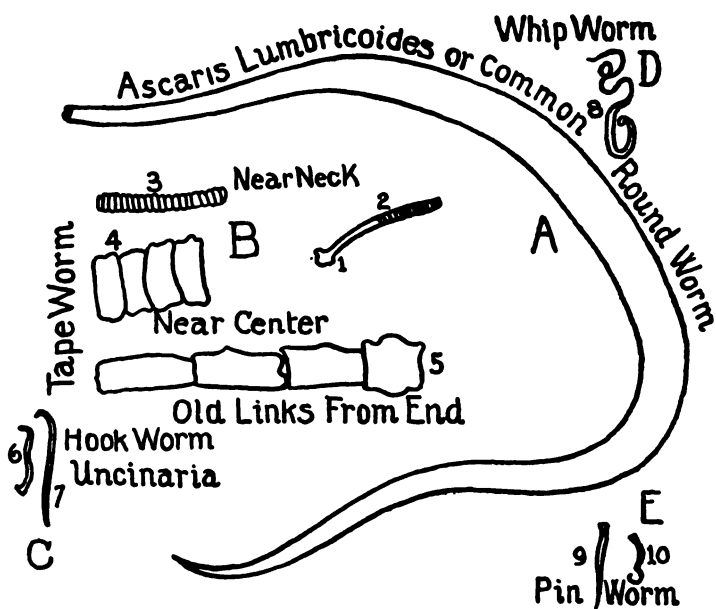


FIG. 113. Some of the larger animal parasites. (All natural size.) A, ascaris lumbricoides or the round worm. B, fragment of a tapeworm. 1, the head; 2, the neck; 3, a fragment near the neck. C, the hookworm, the cause of the anemia of the South. 6, male; 7, female. D, whipworm. E, pinworm; 9, female; 10, male.

The diagnosis is easily made, as the eggs of this parasite are abundant in the stools. The worms are easily killed by a few doses of thymol.

Ascaris Lumbricoides ("Round Worms").—This intestinal parasite (Fig. 113, A) is very common, especially in children. It is from four to twelve inches long and about as thick as a slate pencil. It lives in the small bowel and so long as it stays there seems to do no harm. But when it wanders, it may do much injury. Sometimes it creeps into the stomach and is vomited. Sometimes it gets into the

throat and then into the trachea. From the throat it may get into the middle ear by crawling through the Eustachian tube, then rupture the drum and appear in the external ear. It may wander up into the bile-duct. It may thrust its head through a gastric or typhoid ulcer.

Many symptoms of children—nervousness, twitching, convulsions, etc.—are attributed to these worms, but are probably produced by other causes.

The worms are easily removed by a dose of *santonin* followed by a dose of *calomel*.

Oxyuris Vermicularis.—This intestinal parasite, the “pin-worm,” “threadworm,” or “seatworm,” is very commonly found in the rectum, especially of children. “Pinworms” are little white, thread-like worms, about a quarter of an inch long. They cause intense itching, especially at night. They may do much greater harm than merely to disturb the sleep and should be got rid of. *Santonin* may expel them, but local rectal irrigations with strong salt solution, or quassia water, etc., are most effective (Fig. 113, *E*).

Tapeworms.—The tapeworms are large animal parasites that live in the intestines of men. They are long and consist of many thin, broad segments or “links.” Their name is due to their shape (Fig. 113, *B*).

The BEEF TAPEWORM is the most common tapeworm in America. One gets it from eating insufficiently cooked beef from an animal in whose muscles were many embryos of this worm. The worm grows to a length of fifteen or twenty feet. Its largest links are nearly a quarter of an inch broad, and about half an inch or more long. The head is about the size of a common black-headed pin.

The PORK TAPEWORM is very rare in America. It develops from a living embryo of this worm—an embryo eaten with infected pork. It is smaller than the worm described above, being only from six to twelve feet long, and has somewhat smaller links. Its head also is smaller, but is provided not only with suckers, as is the beef worm, but also with hooks. It is a much harder worm to destroy than the beef worm.

BOTHRIOCEPHALUS LATUS is a tapeworm common in parts of Europe and the far East, but very rare in America. Its embryos are eaten with fish and will infect man if these infected fish are eaten raw. It may grow to a length of from

twenty-five to thirty feet. This worm sometimes causes an anæmia which can hardly be told from primary pernicious anæmia.

The symptoms of the beef and pork worms are few, if any, until the patient knows that he has the worm, and then any number of most distressing nervous symptoms may occur.

The diagnosis of all large tapeworms is easy, for their links are passed in most of the stools and when seen cannot be mistaken. But the opposite mistake is often made, for many patients are sure that the shreds of mucus or of vegetable fibres that they pass are tapeworm links and they explain the ragged, translucent appearance of the "worm" by the supposition that it is "decayed." But the segments of a tapeworm do not easily decay.

One avoids tapeworms by abstaining from ill-cooked beef and pork. In America the meat inspection must become much more rigid before it will be safe to eat the raw meat which so many enjoy.

The treatment consists in first cleaning the bowels well with saline purges, and then giving a large dose of one of several drugs. This dose will either kill the worm or at least stupefy it, so that it will cease to cling to the intestinal wall and will be passed in the stools. Among the remedies used are male fern, pomegranate root, turpentine, and (one of the best) a pumpkin-seed tea, consisting of water in which three or four ounces of crushed pumpkin seeds have been soaked for twelve or fourteen hours. A purge must be given soon after this remedy, to make sure that the worm is hurried out of the bowel. One must always see the head before he can be sure that the cure is accomplished, for, if even twenty feet of worm are passed and the head is left behind, in a few months there will grow from it twenty feet more. The head is easily recognized, for it is a small ball, about the size of the head of a pin, on a thread-like neck. The stool which is expected to contain the worm should be passed into a vessel nearly full of warm water. If the water is cold the worm may vigorously contract and break, and the head be left.

CYSTICERCUS DISEASES. "MEASLES."—*Cysticercus* disease also is an infection with the pork tapeworm. This is

the disease the hog had from which a man gets the adult worm (see above). Man also can get this form if he swallows the eggs. We get the tapeworm by eating the embryos which are encysted in the flesh of an animal. We get the embryos by eating the eggs. Each embryo will develop into an adult worm.

The tail segments, the oldest parts, are each full of eggs. It has been estimated that a tapeworm may "lay" over forty million eggs a year. These eggs, probably through drinking

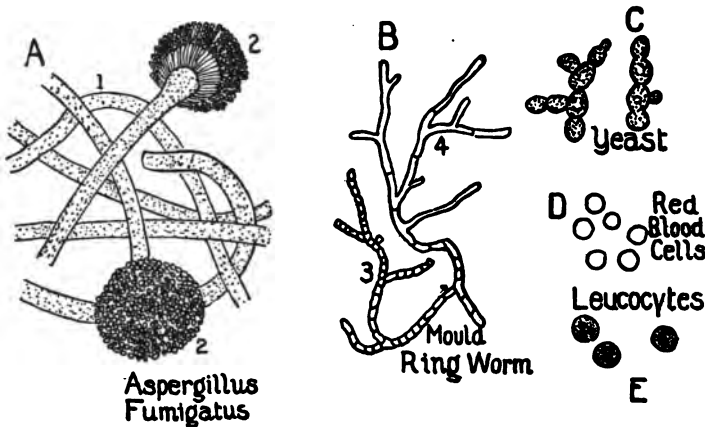


FIG. 114. Some other important vegetable parasites. A, a mould; 1, the mycelial threads, 2, the fruit head. B, the parasite causing ring worm; 3, threads which are dividing into spores from each of which a new "plant" can grow; 4, threads not sporulating. C, a common yeast. D, red blood-corpuscles, and E, leucocytes, introduced for comparison of size. All are drawn to the same scale. Magnified 250 times.

water, reach the stomach of a pig, the "intermediate host." In the pig's stomach the gastric juice digests off the shell, and the egg hatches an embryo, or larva, which makes its way through the wall of the stomach or intestine and is carried by the blood to almost any part of the pig's body. Wherever a larva settles it soon builds a wall around itself, thus forming a small cyst. It then develops no further and causes no more harm. Each cyst is a little bladder from one-quarter to three-quarters of an inch long and about half as big in diameter, and each contains the head of a tapeworm. It is this head which when swallowed by a man develops into the whole worm. The pig's entire body may be studded with these little cysts, each containing a living embryo. The pork from

such an animal is said to be "measled." But if a man swallows the eggs, he then becomes the "intermediate host," and his body becomes "measled." In his muscles and internal organs, and under his skin there may be thousands of these little cysts. The most of them do no harm, but one which develops in the brain or the eye, etc., can do more harm than all the rest together.

TÆNIA ECHINOCOCCUS.—The tapeworms already mentioned are very large worms. There is one tapeworm, *Tænia echinococcus*, which is only about one-fifth of an inch long. This lives in the intestines of dogs, and can cause far more serious disease in man than can any of the other worms. If the eggs of this worm get into the stomach of a man, they hatch, and a tiny embryo is set free. This burrows its way through the intestinal wall and then is carried by the blood to some organ of the body. Wherever it lodges it changes into a little cyst, or bladder, containing the head of the worm. While thus far it resembles the cysticercus of the pork tapeworm, its later development is very different. This little bladder can "bud" and "bud" again, hundreds of times, each bud producing a new cyst just like the first. These "daughter cysts" lie within the original cyst, which now has swollen in size. Each of these "daughter cysts" may in turn bud, and soon contain many "granddaughter cysts." The one original cyst will now be very large and full of hundreds of these small cysts.

The symptoms of this cyst will be those of a tumor and will depend on the organ in which it lies. *Echinococcus* is common in Australia, Iceland, and parts of Europe,—wherever dogs are allowed to live intimately with men. It is rare in America.

The treatment is to remove the cysts by operation.

CHAPTER XVI

THE TEMPERATURE, RESPIRATION, AND PULSE

The temperature of the internal organs of man, one of the "warm-blooded" animals, is in health practically constant, and considerably higher than that of the external air. This heat is generated in the muscles during contractions, and in large glands, in which combustion is very active. If it were not for the blood stream these organs would be much warmer than those in which combustion is less active; but the blood is constantly flowing from them to cooler parts of the body, and cooler blood is always flowing from the latter to them. At the same time there is a continuous loss of heat from the skin and the lungs, and in the secretions (the urine, etc.).

Although the external temperature varies during the year by about 120° , the temperature inside the healthy body does not vary more than a fraction of one degree, because there is at the base of the brain a "heat regulator" centre, which controls matters with wonderful accuracy. When the outer air becomes cooler, the blood-vessels in the skin are made to contract, and so on the surface of the body there is less blood to cool off. This cools the skin and stimulates us to make our muscles contract oftener and with greater force. When we do not under these circumstances contract them voluntarily, they contract involuntarily, and we "shiver" and our teeth chatter. The sensation of cold leads us to put on warmer clothes, to eat more food, and to drink hot fluids. By the first act we diminish heat loss, by the second we increase oxidization, and by the third we add heat directly to our body. When there is a tendency for the body temperature to rise, the skin vessels dilate, so that more blood is where it can cool rapidly; we perspire more, and the evaporation of fluid from the skin cools us considerably. We exercise less, dress more lightly, eat less, take cold drinks, and each of these practices either lessens the heat production or favors its loss.

The body temperature is not at all the same as the skin temperature, which varies greatly. The old method of determining temperature was to put the hand on the skin and decide whether or not the patient "felt feverish." This method is roughly accurate, provided the hand is placed on the skin of a portion of the body protected at the time by clothes, so that there is just then no great heat loss, and provided the patient is not in a chill or sweat. In the case of a chill the skin may actually be colder than normal, and yet the blood temperature high; in the latter case the skin may be warmer than normal, and yet the blood temperature normal. The "blood" or "body" temperature is determined by a thermometer placed in the mouth, the axilla, or the rectum. Each method has its advantages.

In America the mouth temperature is oftenest taken. The thermometer is placed under the tongue, the lips are tightly closed, and the thermometer is left in the mouth until the mercury reaches a constant point. How long this will take depends on the pattern of the thermometer. With very ill patients or patients at all toxic this method is not satisfactory, because they will not keep the lips tightly closed. Another disadvantage is the danger of infecting the patient with a thermometer just used in a case of infectious mouth disease, and used before it is thoroughly cleaned.

The axillary temperature is easily and more hygienically obtained, but to take it requires a much longer time. The nurse must be sure that the arm is held close to the body.

For very ill patients the rectal temperature is to be preferred, as nothing is required of the patient. The same precautions against infection must be used as in the case of the mouth. The nurse must also be sure that the rectum is perfectly clear of any fecal masses, no matter how small; she can have this certainty only by washing the rectum out with an enema just before inserting the thermometer. The reason for this precaution is that heat is generated in the feces, as any one can infer who looks at a manure pile on a cold day. The bacteria of the colon decompose—that is, "burn up"—all the organic matter of the stools which has escaped absorption above, and the temperature of this fecal mass is that of the intestinal wall plus the degree or so added by this local combustion.

Of these three temperatures, each taken with the greatest care, the rectal will be a trifle the highest,—about half a degree—as it is venous blood from the bowel mucosa, one of the heat-producing organs, that warms the thermometer. If this is highest by a degree or so, one may always suspect fecal masses. The axillary temperature is apt to be a trifle the lowest. The temperature should always be charted, for the shape of the curve is important. It is taken once in two or four hours, until practically normal, after which it is taken only at 8 A.M. and 4 P.M.

The normal mouth temperature is about 98.7° F. While, for corresponding hours the body temperature scarcely varies from day to day, yet from hour to hour of the same day it does vary, and often by as much as a degree. The curve is lowest between 2 and 6 A.M. The curve rises slowly until about 10 A.M., then drops slightly, then rises towards its highest point, which it reaches between 4 and 8 P.M. In some normal persons this normal curve has quite pronounced rises and falls. A variation of two full degrees Fahrenheit during the day cannot be considered normal; a rise to 99° F., or at least to 99.5°, is to be considered abnormal or "febrile." Theoretically "fever" includes more than an elevation of temperature; practically it does not. For the normal temperature to rise to 99° F. each afternoon or evening, especially the latter, shows some abnormal condition; in persons who otherwise seem well this trouble is most often latent tuberculosis. Temperatures below 98° are "subnormal." "Hyperpyrexia" is the term used for those over 105° F.

The most common causes of fever are the toxins of bacteria; but in exophthalmic goitre, anæmia, leukæmia, or during the time when blood is being absorbed after hemorrhage, there may be a fever that is not due to bacteria.

While these toxins of germs may be called the "cause" of the elevation of temperature, yet the word "reason" is better. Fever seems to be one of the protective measures of the body, a means to an end, and that end is a fight against the germ. The body seems to fight the germ better at a higher than at normal temperature, and so the fever is really rather a part of the defence than a direct result of the toxin in the sense in which the headache is such a result. We

emphasize this point because so many persons think the fever an evil and try to lower it by drugs. They can do this easily, but with no benefit—perhaps always with injury—to the patient. We like to see the temperature fall, as after a cold bath, but only when we are sure that the reason for the fall is victory over the toxin, or that the total result of the bath is beneficial. Of course the temperature may be dangerously high, and then it must be reduced by any means possible. A sunstroke, for instance, brought about by hard work in hot, moist air, is the result of the entire break-down of the thermo-regulator centre, and in such a case the fever may rise to from 109° to 112° F. One combats it with a continuous cold bath, sponge-baths of ice-water, ice packs, ice-water enemata, etc. Some cases, however, fall as if struck down and die at once.

It may be said, in general, that the height of temperature is really no index of the severity of the case. The highest temperatures occur in the least serious fevers, such as relapsing fever and malaria, while in the severest fevers, the rapidly fatal infections, there may be no rise of temperature at all. In the latter cases it seems as if the body were unable to make a febrile defence against the infection.

The type of fever is judged from the highest and lowest points of the temperature of one day. The fever is said to be "constant" when these points do not differ by more than 2° F.; "remittent" when they differ by not less than 3° F., but yet the curve does not touch normal; "intermittent" when the low point is at or below normal. The temperature may rise suddenly, usually with a chill, as in malaria, pneumonia, scarlet fever, and most of the fevers that occur during childhood; or slowly,—that is, during two or more days,—as in typhoid. The best examples of constant fever are lobar pneumonia, and typhoid fever during the end of the first week and the beginning of the second. The temperature may fall suddenly, usually with a sweat, as in malaria, pneumonia, et al.; or slowly during several days. If the temperature falls from a high point to normal within twelve hours, the fall is called a "crisis;" if within twenty-four hours, it is called a "prolonged crisis;" if within more than twenty-four hours, it is said to fall by "lysis." The temperature curves of some diseases are very distinctive and will often suggest the diagnosis.

The Respirations.—The nurse should count the respirations while the patient is unaware of what she is doing, for if he is conscious of her action the rate will almost certainly change. The easiest way is to pretend to be counting the pulse. The respirations are counted for at least half a minute, better for a whole minute. The normal rate for an adult is from 16 to 24 per minute; for a child about 20, for an infant about 44.

A person inhales by increasing the capacity of the chest. This is done by raising the ribs (costal breathing) or by lowering the diaphragm (abdominal breathing) or by both. When the patient will not expand his chest we suspect pleurisy, etc.; or we may find that the ribs have become solidly united to the backbone, as occurs in arthritis deformans. When the patient will not "lift his abdomen,"—that is, will not breathe with his diaphragm,—we suspect peritonitis. The patient may move the ribs on one side of the chest more than on the other. This may help us to tell on which side the pneumonia, pleurisy, or tuberculosis is, for that side will expand less.

Increased rapidity of respiration may be due to various causes. Some nervous persons will breathe even sixty times a minute, but the respirations are full, easy, and painless, and hence we cannot say that the patient has dyspnoea. He has "polypnoea." Other nervous patients breathe slower than is normal; these have "oligopnoea." If a person every minute or so takes a long, sighing breath, there is good evidence that he is neurotic.

When respiration is, for any reason, difficult and therefore requires unusual effort, the condition is named "dyspnoea;" if the patient cannot lie down, but must sit up, in order to breathe, his condition is "orthopnoea." Sometimes pain makes the patient breathe more rapidly than is normal. Such is the case in pleurisy; it is painful to move the chest wall much, and so the patient takes short, quick breaths. This is seen also in neuralgia of the chest, in "rheumatism," trichiniasis, et al. If for any reason the amount of breathing lung is diminished, the person will breathe more rapidly with the amount left. This is seen in consumption, in pneumonia, in cases of pleural effusion, in which fluid has allowed the lung to collapse, and in cases of pneumothorax, in which air

has produced the same effect. Dyspnœa may be the result of a fault in the circulation, not in the lungs. The rate of respiration is controlled in the brain, and one condition which stimulates the "respiratory centre" is an increased amount of carbonic acid in the blood. This is present when there is poor circulation through the lungs, as in pulmonary emphysema, and especially in heart disease, but also when there is not enough hæmoglobin to carry oxygen to the tissues, as in anæmia. The dyspnœa of heart disease—"cardiac asthma"—is especially distressing. In some conditions the patient breathes slower than is normal, but each breath is a long, deep one, requiring a struggle. Such is the case in asthma; here the air enters the lungs more easily than it leaves, and so expiration is prolonged. In bronchitis, with the little tubes almost closed, the respiration may be quite rapid; or, rarely, it becomes slower, and the "rattling" of the bubbles of exudate in the bronchi may be audible all over the room. In other cases the struggle is to get the air into the lungs, as in a case of obstruction at the larynx from œdema or diphtheria ("membranous croup"), or when a foreign body has lodged in the wind pipe.

One speaks of "apnœa" when respiration ceases entirely for a few seconds. People are always apnœic after taking several long breaths, for these breaths have provided a surplus of oxygen, and respiration will not begin again until the carbon dioxide has reaccumulated in the blood. The best illustration of apnœa is seen in Cheyne-Stokes breathing. The patient lies quiet for perhaps fifteen, twenty, or even forty seconds, without taking a breath. Then he begins breathing, very gently at first, then deeper and deeper, until he sits up in the most intense orthopnœa, and struggles even in drawing very long, rapid breaths. Then these become quieter and quieter until apnœa again begins. Cheyne-Stokes breathing is most often seen in cases of arteriosclerosis, chronic heart disease, and Bright's disease. In these cases it is a serious, but not an especially threatening symptom. It occurs in meningitis and after injuries to the skull, or with coma, and then it means serious danger.

The Pulse.—Every beat of the normally functioning heart sends a wave along the arteries to the capillaries. This wave is felt as a pulsation of the artery's wall and is called

the "pulse." We count the pulse as an easy way of counting the rate of heart-beat. Usually the pulse is counted at the wrist, on the radial artery. This vessel is convenient, lying, as it does, just under the skin, and resting directly on a bone, which serves as a solid foundation for it. We might, of course, choose any artery, but should always use the same one, as there is a great deal in "getting used" to a vessel. Only when this is done can one appreciate minor differences. To count the pulse, we place three fingers of one hand on the artery and then exert just that pressure which will make the waves most distinct. One does not count with the thumb, as that has a pulse of its own, and the observer might unconsciously count his own pulse. Three (or at least two) fingers are used because one thus gets the most accurate idea of not only the number but also the character of the pulse waves. One first feels the pulse for a short time, in order to gain an idea of what sort it is, and then counts the number of beats felt in fifteen seconds. This number multiplied by four is the pulse-rate per minute.

One first notes the *regularity of the pulse*. The beats of a normal pulse are of almost equal force and are separated by intervals of almost equal length. In some cases, as those with disease of the mitral valve, and, more especially, those with myocarditis, the beats are irregular in "force and rhythm." That is, some beats are strong, some weaker, and some are scarcely felt at all; some follow one another in rapid succession, while the interval of time between others is longer. In such a case one is careful to specify that he is counting the pulse-rate, not the heart-rate, for he may be certain that the longer pauses are filled with small beats which are too feeble to be felt at the wrist. To judge the rate of such a heart-beat, one must listen to it directly. Vessel walls which are very inelastic easily obliterate the feeble beats; an aneurism on an artery, or the pressure of some neighboring organ or tumor, also can prevent beats from passing through. The result may be that the man "has two pulses;" that is, feeble beats can be counted at one wrist but not at the other. In some myocarditis cases the heart is so irregular that we speak of its condition as "delirium of the heart" (delirium cordis).

In some cases the beats come "in twos,"—the "bigeminal"

pulse. This is not uncommon in a patient to whom has been given a little too much digitalis. Or the beats are in threes,—the “trigeminal” pulse. In some cases it seems as if a beat were occasionally dropped. As a rule, however, there is a heart beat, but it closely follows another beat and is itself weak. The next beat is occasionally very strong indeed. This often occurs in persons apparently healthy. They feel each of these “extra systoles,” as they are called, and become very nervous about their condition. They are sure they have heart disease. Some say that their heart “jumps,” others that it “stops.” Some of these are merely nervous persons; in some the unpleasant experience follows the use of tobacco, of coffee, tea, etc.; in some it is caused by indigestion; some have real heart disease, in which extra systoles are common enough. Sometimes each second beat is very feeble while the others are strong. These feeble waves may be real pulse waves, due to feeble beats. In some cases of digitalis poisoning this pulse is found; in others all these feeble beats drop out,—that is, are not felt at the wrist—and we count only half the true heart beats. The doctor thinks the digitalis has slowed the heart beautifully, whereas really the heart is poisoned and is beating twice as fast as he thinks. But more often one feels a pulse like this when there is a markedly “dicrotic” wave, such as occurs in typhoid fever; that is, the feeble wave is only a part of one pulse beat. Unless careful, one will count this pulse as twice as frequent as it really is.

The next point one notices is the *pulse-rate*. In the normal man the rate is about 72 beats per minute; in the normal woman about 80. The rate depends somewhat on the position of the patient. For instance, the pulse-rate of a normal man is about 66 when he is lying down, 71 when he is sitting, 81 when he is standing, and even more when he is walking. This is why we insist that patients with heart troubles shall lie flat as much as possible; they thus save the heart about 15 beats a minute, 900 an hour, 21,600 a day. The rate depends on the age also. At birth the pulse-rate of the normal child is from 124 to 144. It becomes gradually slower until puberty, when the normal rate is from 72 to 92.

The rapidity of the heart is controlled at the base of the brain, through the tenth or vagus nerve. Cut this nerve,

and the heart will continue to beat, but only 26 or 28 times a minute. In the Stokes-Adams disease this is about the rate. The vagus is not cut, but a little band of muscle fibres, the "wire" over which the contraction passes from the auricle, the usual starting-point of a contraction, to the ventricle, is so injured by disease that few stimuli pass through. The result is that the auricle continues under control of the brain and beats almost the normal number of times; but the ventricle is, in an extreme case, entirely independent and so beats 26 or 28 times a minute. In other cases a few of the stimuli do pass, so that the rate of the ventricular pulsation, and hence of the wrist, is 28 plus the number of the stimuli that pass from the auricle. In this disease we can see the veins pulsating more than seventy times a minute—since the pulse of the veins comes from the right auricle—and can count a very slow pulse at the wrist. Patients with the Stokes-Adams disease have frequent fainting spells.

A multitude of causes can increase the heart rate. When this is very high, the condition is called "tachycardia." Among these causes are hard muscular work; mental influences, as emotions, surprise, fear, delight, etc.; alcohol, coffee, fever. An increase in heart rate is as much a symptom of fever as is the rise of temperature, while the pulse-rate in fever is of even greater value for prognosis than is the temperature. No matter how high the temperature, one does not worry so long as the pulse is fairly low; but let the heart rate rise to 140 or over, and the outlook becomes at once more serious. As a rule, in a strong man the pulse-rate will increase about five beats per minute for each degree of rise in temperature. In some fevers the rate is increased more than this, as in pneumonia especially and in fevers with rapid respiration. In other high fevers the pulse-rate may rise scarcely at all. This is true of typhoid fever, and often of meningitis, while in yellow fever the pulse becomes slower as the temperature rises.

During a break in cardiac compensation the pulse is nearly always rapid. In exophthalmic goitre a rather rapid pulse is one of the most important symptoms. A rapid pulse and very low temperature occur in "shock" or "collapse," due, for instance, to accident, to severe hemorrhage, to peritonitis, etc. Some persons have attacks of "paroxysmal tachy-

cardia." That is, the heart suddenly begins to beat fast, about 160 per minute, often exactly twice or exactly three times as fast as before; then in a few minutes, hours, or days, it suddenly returns to its former rate. These attacks often occur in cases of myocarditis without the patient's knowing anything about them; but when these paroxysms occur, as they sometimes do at intervals for years, in an apparently healthy nervous person, he is usually painfully aware of them.

In "bradycardia," or an abnormally slow pulse-rate, one must be sure that the heart rate really is slow and that there is not a loss of many feeble beats. The most common cause is jaundice, for the bile in the blood poisons the heart. It occurs also during the convalescence of fevers, typhoid especially, but also of many others, and in yellow fever during the attack. The pulse is chronically slow in myxœdema; in all conditions increasing the intracerebral pressure, as a fracture or brain hemorrhage; in Stokes-Adams disease; when the heart is poisoned by digitalis, sometimes by coffee or tobacco; in many chronic debilitating diseases, etc.

The next point to notice is the *tension of the pulse*,—that is, the amount of pressure we must use to prevent the pulse-wave from going any further in the artery. We press with one finger and feel for a wave beyond that finger with the others. As a rule, we can easily close the artery, but in cases of arteriosclerosis and of Bright's disease we may press very hard and yet beats will pass through. But we may be deceived. Sometimes there is an unusually large branch connecting the radial and ulnar arteries below the point observed. Then press the radial as hard as one will, and a wave is felt beyond, but this wave "comes back" and does not pass under the pressing finger.

Then we notice the *character* of the pulse. It may be "big" and "bounding" or "small" and "feeble." It may be "quick," as in aortic insufficiency. In such a case the waves strike the fingers with a quick, sharp stroke of surprising force. One can feel the pulse even at the fingers' tips, can see the flesh under the finger-nail blush, etc., with each beat (the "capillary pulse"). But this same pulse is felt in severe anæmias, in some nervous persons, and in almost any normal person after a warm bath.

Sometimes the heart-beats are feeble, and the pulse-waves

are scarcely felt or even not felt at all. This occurs near death from any cause, in some cases of mitral heart disease, and in Addison's disease. A feeble rapid pulse is a serious sign, but when the pulse is feeble and not rapid one should carefully examine the wrist, for a person with such a pulse may have an unusually small radial artery. In some persons this artery runs down the side rather than the front of the wrist, and the vessel may exhibit other abnormalities that might deceive us in judging of the pulse. For that reason we should always feel both wrists and then, if necessary, other arteries, especially the facial. In aortic stenosis, a condition in which the blood is slowly squeezed into the aorta through a small hole, the character of the pulse is, as we would expect, not "quick," but "slow." This has relation not to rate, but to the speed with which the wave reaches its crest. Sometimes the dicrotic wave is distinctly felt. In a case of fever this wave suggests typhoid, but it is common enough in other fevers.

And, lastly, we judge of the *thickness of the vessel wall*. We first close the artery with one finger and then with the other fingers feel the empty vessel. The normal radial artery of a young person cannot be felt through the skin, but with increasing age it becomes thicker and stiffer, so that it feels like a rubber tube; and when the arteriosclerosis is extreme, it feels like a "pipe-stem." Then not only is it uniformly thickened, but in the walls are solid plates which make it feel like a "goose's neck." Arteries which are sclerotic are usually tortuous also and may appear like a snake under the skin. (The temporal artery is normally always snake-like.) The blood in them is, as a rule, under high tension.

CHAPTER XVII

SIGNS AND SYMPTOMS

Observation of Symptoms.—With a confidence not formerly felt, the trained nurse is now entrusted with the care of the patient for most of the day and is held responsible for the correct observation and recording of the important features of the case. She is supposed to have experience as well as knowledge and to be able to recognize each disease's danger signals, and these, for the most part, can be read only by the trained eye.

The mental condition of the patient should always be watched. While marked abnormalities are easily recognized, one may easily fail to detect their first traces, which indicate the time to begin active therapy or to guard the patient or his friends.

Among the abnormal mental conditions are the following: "apathy" or mere indifference, well illustrated in nearly all typhoid patients; the "twilight" condition in which the patient is "half-awake," often delirious when quiet, fairly easy to arouse; "somnolence," which renders the patient abnormally sleepy, and which is sometimes the first sign of oncoming diabetic or uræmic coma; "stupor," from which he can only with difficulty be aroused; and "coma," from which he cannot be aroused.

Conditions of mental confusion are to be observed. In various mental states the patient is not "orientiert" as to time, place, persons, etc. That is, he is not sure where he is, how he came there; he is doubtful as to the time of day, or the day of the week, etc.

Unusual loquaciousness, mental exaltation, or the reverse—depression of spirits—are often important signs. Any suggestion of ideas of persecution, a distrust of surroundings, and delirium in a patient without fever should put the nurse on her guard.

All the features mentioned above may be, but are not

always, signs of actual mental disease. We now turn to features present in persons of normal mentality.

Near the beginning of this chapter we would emphasize the difference between the signs and the symptoms of a disease. The symptoms are those features of the patient's condition of which he himself complains. The signs are evident to the doctor also. The latter he can see, or hear, or feel. For instance, a patient complains that every time he takes a breath he has a pain in his side, and therefore he draws only rapid, quick, short breaths. This pain is a symptom. We have at first only his statement to lead us to believe that he has pain. We see the character of his respirations and suppose that he is not trying to deceive us, but he might be doing so. Even if we were convinced that it was pain that made him take such short breaths, still we should be in doubt as to the origin of this pain. He might have pleurisy, or rheumatism of the chest wall, or neuralgia of the intercostal nerves, or he might only imagine the pain. The doctor listens to the chest and hears a faint scratchy sound. This scratchy sound is a sign and we are sure he has pleurisy.

Now, symptoms are really poor data in judging of the nature or the severity of a disease, and yet it is they that trouble the patient, and, no matter what his condition may be, when they disappear he calls himself well. Herein lies the success of quacks and of dealers in patent medicines. They cure symptoms, and their patients are satisfied. And herein lies the failure of many of the best-trained doctors. They concentrate their attention on the disease causing the symptoms, and try by curing it to save the patient from future trouble; but if they do not soon relieve the symptoms, their efforts are neither understood nor appreciated, and the patient will employ a doctor that makes him comfortable near the beginning of treatment. Later, when the disease is more advanced and the symptoms cannot be controlled, he may, or may not, regret that he changed doctors. To make a patient comfortable and to cure his disease are two separate problems, and the doctor should be an expert in both.

But, while signs are the province of the doctor, symptoms are in a peculiar way that of the nurse, and she should apply herself to learning those innumerable little "tricks" of her profession by which patients are made comfortable, remem-

bering that no symptom is too trivial to be disregarded if it troubles the patient, and that the skilful application of simple nursing methods can transform a complaining sufferer to a contented and fairly comfortable patient.

The public, long educated by the advertisements of various proprietary medicines—advertisements that emphasize abnormal feelings—has been very slow to believe that there can be serious disease without symptoms. And yet this is true of some serious diseases for the most of their course, and of a larger number of diseases during some part of it, especially the early part, while they are still curable. The small cancer that will later kill the patient is often unobserved for a long time. Many a man learns for the first time when he applies for life insurance that he has heart disease or advanced Bright's disease, which thus far has been entirely without symptoms, but which later will be only too evident. A man may lie dangerously ill of typhoid fever or tuberculosis, and yet be happy because "he never felt better." Some man who died from accident is found at autopsy to have had an aneurism which he never suspected, or a gastric ulcer almost ready to perforate the stomach wall. Another man, who never knew he had a serious heart trouble, falls dead of angina pectoris. Many more illustrations might be given, but these are enough to show how hopelessly at variance are our feelings and our condition.

Many serious diseases make their presence known to the patient chiefly through disturbances of organs other than the ones diseased, disturbances which can be cured only by relieving the primary disease. Here is a patient who sits upright in bed in his effort to breathe. He has a distressing cough and expectorates much sputum, and also blood in considerable amounts. Examine his lungs, and a severe congestion and bronchitis are found. He is sure that he has a serious lung trouble, but he really has a heart trouble and doesn't know it. The lung congestion and bronchitis would clear up in a few hours, could we cure his heart. Here is a patient with "rheumatism of the feet;" they are swollen and sore. Or he may have trouble at the opposite extremity of his body,—a terrible headache, and hemorrhages into his eyeballs. Or his trouble may be exactly between these two extremities of his anatomy,—in his stomach,—and any one

seeing his attacks of vomiting will believe that there lies the trouble. But neither his feet, head, nor stomach is diseased; his real disease is in his kidneys, organs which have not themselves given one single symptom. Here is a man who knows he has hemorrhoids, and who also has almost died from profuse hemorrhages from his stomach. He does not know that a serious liver disease is at the bottom of both these conditions.

Sometimes the primary trouble is much less serious than those which it causes. This patient has chronic laryngitis and bronchitis, but it is possibly an unsuspected suppuration in the nose which originally caused these troubles, and which now keeps them alive through the pus which constantly drips down into the throat. Here is a dying man. He has had repeated attacks of that most terrible disease, acute endocarditis, which have injured his heart, each more than its immediate predecessor. Now he is in a pitiful condition and cannot live long. This man in the next bed has had repeated attacks of inflammatory rheumatism, and is now a helpless cripple. But the primary trouble in both these cases may have been a simple chronic tonsillitis, which they do not know they have had. Had that been cured months or years ago, the chances are that these men would now be free from the diseases that afflict them.

In a much larger group of cases the organ which gives rise to the symptoms is not, so far as we can see, diseased, although its function certainly is disturbed. The symptoms of which the patient complains are signals of trouble in some other, perhaps distant, organ. The organ producing the symptoms resembles the semaphore seen beside the railroad track. Its arm is raised, or the red light is shown, and the engineer of the approaching train stops the engine, not because he thinks there is anything wrong with the semaphore, but because it indicates trouble farther on, perhaps half a mile down the track. So there are persons who have the most distressing attacks of asthma,—attacks which may lead to permanent and serious lung trouble,—and yet these attacks are sometimes only signals indicating adenoids in the roof of the pharynx, or a polyp in the nose. Here is a girl with troublesome attacks of nasal obstruction, but the swelling of the mucous membrane of her nose is perhaps only a signal

of trouble in her pelvis. This child has had epileptic attacks, and yet they may possibly stop if proper attention is paid to the eyes or to some other organ. This person complains of headache, sleeplessness, loss of memory, inability to do mental work, but all these symptoms disappear when his constipation is cured. As a final illustration we give two of the most common experiences. Some dyspeptic patients may for weeks complain of no symptoms but those from the stomach,—utter loss of appetite, gastric distress, and frequent vomiting. We may treat the gastric condition, but without success. The trouble is a little unsuspected patch of tuberculosis in the apex of one lung. The gastric symptoms are the signals of danger (in the lung) displayed at a distance, in an organ (the stomach) not itself diseased. In chronic appendicitis, again, it may be the stomach that seems to be the suffering member.

Finally, we wish, in this connection, to speak, not of symptoms of definite disease in healthy organs, but of referred pains,—severe pains distant from the seat of the disease. In gall-stone colic much of the pain may be in the shoulder; in acute inflammation of the gall-bladder the pain may all be in the left side, a foot away from that organ; in pelvic trouble the pain is sometimes in the back or the head; in incipient hip disease the pain is in the knee; in heart disease the pain may be down the left arm; etc.

We have attempted to show how easily one may be deceived if he depends on symptoms. If one searches for signs with seeing eye, he will, in practically all such cases as those cited, correctly locate the real trouble.

Of all symptoms pain is the one which interests patients the most. We here emphasize the truth, too little understood, that pain is an unpleasant sensation, nothing more, and is *never* imagined. Imagination may be its cause, but the pain thus produced hurts just as truly as pain produced by a real disease. Pain is only a phenomenon of consciousness; it is *always* real, even that felt in a dream. If the patient is too unconscious to feel it, there simply is no pain, no matter how badly the person's body may be injured.

Pain is always mental and never is in the place where we think we feel it. A common illustration may help. Suppose that (Fig. 115) A is the home of Mr. Smith, of Baltimore, and

that his telephone is connected through a central switch-board with other telephones—*B, C, D, E, F*, etc. Mr. Smith has but one instrument. The bell rings. He takes down the receiver, answers the “Hullo,” and asks, “Who is it?” For if the voice is unfamiliar, he has no idea who or where the stranger is. This person may be at any of the nineteen thousand or more instruments in Baltimore, or at any one of those in the towns around, or even in a distant city. The same receiver serves Mr. Smith for all, the same diaphragm of iron by vibrating renders audible all the messages that come over the wire to his ear, and Mr. Smith answers all by speaking into the same transmitter. The stranger gives his name and his address. Then Mr. Smith can “locate” the speaker. But Mr. Smith does not always ask, “Who is it?” He often recognizes at the first word a familiar voice, and is almost sure that he knows where his friend is. He then seems to talk with his friend directly, as if in the same room with him. He can almost see his friend as he listens to his voice. Or the person who has called him up gives no name, but has something to say about a grocery order, or refers to the contents of a letter which Mr. Smith wrote yesterday. Mr. Smith at once knows the firm whose representative is speaking. But, whatever the message, whoever or wherever the speaker, Mr. Smith hears only his own receiver vibrating. And yet he often says, “I recognized the voice at once.” Of course he did not hear his friend’s voice. Perhaps miles of wire separated him and that friend whose voice was reproduced by his one receiver. While he is often fairly certain of the voice, though perhaps never positive, he is never sure of his friend’s exact location and must believe he is just where he says he is, for the voice will sound the same at any

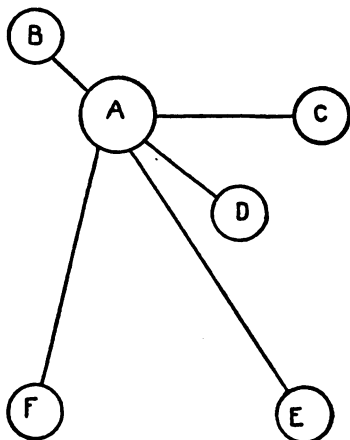


FIG. 115. Scheme of a telephone system and of our sense organs. *A*, central station. *B, C, D, E, and F*, various call boxes.

but has something to say about a grocery order, or refers to the contents of a letter which Mr. Smith wrote yesterday. Mr. Smith at once knows the firm whose representative is speaking. But, whatever the message, whoever or wherever the speaker, Mr. Smith hears only his own receiver vibrating. And yet he often says, “I recognized the voice at once.” Of course he did not hear his friend’s voice. Perhaps miles of wire separated him and that friend whose voice was reproduced by his one receiver. While he is often fairly certain of the voice, though perhaps never positive, he is never sure of his friend’s exact location and must believe he is just where he says he is, for the voice will sound the same at any

of thousands of instruments. Now suppose that the wire is down. The friend may call for hours, but Mr. Smith suspects nothing. Or perhaps Mr. Smith is asleep. The bell rings, the instrument is in perfect order, but Mr. Smith pays no attention, no matter how important the message may be.

Let us now change the figure (Fig. 115). *A* is the "sensorium," the seat of consciousness in our brain, which is tightly enclosed in the skull. The brain is connected with the various sense organs by nerves. One of these sense organs receives a stimulus and sends a nervous current to the brain. There this nervous current is in some way "reproduced" into, or at least transformed to, a sensation. Whether this sensation is pain, touch, heat, light, sound, or some other sensation depends on the sense organ from which it came. For instance, I burn my finger, *C*. The endings of the sensory nerves at that place send a current to *A*, and my mind "feels" the pain. I do not feel the burn any more truly than Mr. Smith "hears his friend's voice." Two feet of nerve separate that burned finger and my brain. I feel the sensation into which my sensorium transforms the nerve current. If the nerve is cut I feel no pain; the mechanism of my sensorium remains mute, although the finger was burned and my mind was capable of realizing it. Or I am stupefied by ether, and I feel no pain. The nervous currents go to the brain, the brain receives those nerve currents, but the mind "has a deaf ear to the telephone."

In the case of the telephone the person calling us up tells his name and number if we ask; in the case of the sensorium we cannot ask, and if we do not "know the voice" of the burned finger, we cannot, though we feel pain, tell the nature or perhaps the location of the injury. The young child who does not yet know the voice of his sensations does what telegraph operators do in time of war. He sends his messages over more than one wire, and verifies the answers. This is why the child, when his finger pains him, looks at it to see if it is red, and touches it to see if that touch increases the pain. A very young child seems to recognize but few sensations. Perhaps he does not know, if we burn his finger, whether he feels, sees, or tastes that burn. But slowly he "learns the voice" of his sensations and becomes able to distinguish his visions from his tastes, his tastes from sounds,

etc. Later he learns to localize these sensations and knows whether the "hurt" came from his toe or his finger.

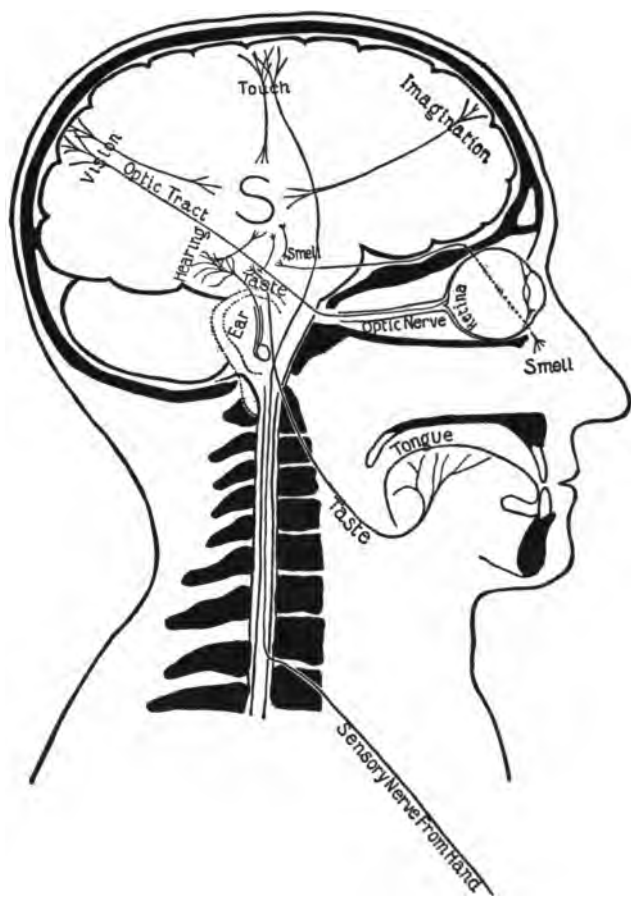


FIG. 116. Scheme of our sense organs. *S*, the sensorium where sensations are perceived (an imaginary spot). Note that stimuli from our sense-organs travel along nerves to the cortex and then along other fibres to a point, *S*, where they are perceived as sensations. We do not "see" with our eye nor "hear" with our ear, nor "feel" with our fingers etc. From the eye, the ear, the finger, etc., start nerve currents which travel to the brain. Here, if the current be strong enough, it stimulates the brain to "see" to "hear" or to "feel."

The adult can seldom be fooled about common sensations from organs which frequently send messages, but even an adult can be badly fooled about the origin of sensations from

those organs in which sensations seldom originate. For instance, a woman has a pain in the right side. Did that pain originate in the muscle wall, the nerves there, the kidney, the gall-bladder, the gall-ducts, the liver, the appendix, or some other organ? The question is sometimes very difficult. We press at various points, asking each time, "Does this cause the same pain?" If we are not able to reproduce it, we ask the gynæcologist to pass a long catheter through the bladder and ureter, into the kidney itself. A little fluid is injected, and the woman cries out. Again we ask, "Is that the same pain?" and from her answer we know whether her original pain was or was not due to an increased tension of urine in the pelvis of the kidney (hydronephrosis).

By "localizing" pain we mean "projecting" it. We feel the burn in our brain, but by our imagination we "project" it to the burned finger, much as Mr. Smith imagines himself talking directly with his friend, oblivious of the miles of wire between them.

In the case of the many "referred pains," and by these we mean pains felt at some distance from the disease, the trouble probably lies in the "switch-board" of the nervous system. We do not "know the voice" of some sensation, and so we cannot locate it. But we do our best, and we project it, often wrongly, to that part of the body from which we are accustomed to get similar sensations, and which "uses, in part at least, the same wires" as does that internal organ from which came the sensation.

There is one "number" at our nerve-telephone-exchange which is always "calling up" but which seldom gives its name. We refer to the brain cortex, where are stored up the memories of all our past sensations. This can "imitate the voice" of eye, ear, skin, etc., to perfection. It does this in a dream. Also, we sometimes say, "I thought I heard you speak," and, when you say you didn't, we say, "I must have imagined it." What we should say is, "I did hear you speak. The voice I heard was just as truly your voice as that which I now hear, but the nerve current causing the sensation of your voice came, not from my ear, but from my cerebral cortex, where are stored up many records of your voice." Dreams, hallucinations, etc., are sensations from the cortex.

Of greater importance than these sensations which arise

in the cortex is that element which the cortex adds to practically all of our sensations. In the sensations which we experience every minute we do not see, hear, or feel exactly what we say we do. We evidently get skeletons of sensations and fill in the details with our imagination and memory. I glance at you and get the picture of your face, as a whole, but a glance of the same duration at any stranger's face would give me very little detail. My memory fills in the details of your face.

The details of the symptoms of disease are provided chiefly by the cortex. A man has a pain in his abdomen, and some one suggests appendicitis. At once the pain assumes a certain character; it resembles the pain which some man with appendicitis once described to him. He is sure his condition is serious, and he certainly feels ill. The doctor is called in and tells him that his pain is on the wrong side. From this time on he has only a simple colic.

But often it is not the quality, but the location, of the pain that the cortex supplies. "Phantom limbs" are a good illustration of this. A man has his arm cut off. Often the cut end of the nerve is irritated and sends currents to the brain. For years every nervous current travelling over that nerve started from the hand and arm and was projected back to them, so that it is no wonder that the mind projects to exactly the same spot the sensations coming over this same nerve, and that the man distinctly "feels" his arm. This point has many practical bearings. For instance, facial neuralgia is due to inflammation in the nerve itself or its ganglia. The patient feels the pain in the areas of skin from which that nerve comes, because his mind projects them there. Sometimes after the nerve is cut the patient complains of the same pain in the skin. We laugh and say, "It is impossible for you to have pain in the skin, for the nerve is cut; you imagine it." The patient might answer, "The pain never was really in the skin; it was in the nerve running from that skin. I mentally projected it to that skin because the skin was formerly the source of all the sensations which ran to the brain along that nerve. If, now, nervous currents still run to the brain from the stump of that cut nerve, is it not natural for me to project the pain to that same area of skin? And does imagination in this case play a more promi-

nent part than in the case of the normal sensations of a healthy person?"

Neurasthenia.—In the preceding paragraphs we have spoken of symptoms, especially pain, as an introduction to a discussion of neurasthenia, which in its pure form may be described as a nervous fatigue with the symptoms, but none of the signs, of organic disease. The symptoms may be those of the most distressing disease of any kind.

This is a most important disease for nurses to study, because its prevalence in this country gives it the name "the American disease," because to the nurse is due the credit of most of our cures of neurasthenia, and because to understand a case of it means that one has a rather broad knowledge of the nursing of all diseases.

Neurasthenia does not shorten a man's life; in fact, it lengthens it, for no one takes better care of himself or demands better care from others than does the neurasthenic. The neurasthenic never dies from his malady, but whole families are literally sacrificed to it, for he demands their care and consideration with a jealousy almost insane. Hence every neurasthenic cured is a family released. For each neurasthenic cured some one deserves much credit. Give one hundred persons ill with typhoid fever only simple nursing, without medical attention, and *vis medicatrix naturæ* will cure at least eighty-five per cent. of them. For these cures no doctor can claim any credit. But submit one hundred cases of neurasthenia to simple nursing, and very few, if any, will recover, while the majority will become worse. Typhoid incapacitates a man for a few weeks; neurasthenia often for a lifetime. The typhoid patient dies but once, the neurasthenics "die daily," and may suffer with an intensity actually not equalled in any other disease.

A case of pure neurasthenia may be described as a nervous bankrupt who pays attention to some trivial sensations which any normal person might have, believes them to be important, and worries over them. The chances are that nervous exhaustion due to any one of many conditions—business, for example—prepared the way. His worry about his symptoms is now the cause of further exhaustion, and this *circulus viciosus* exhausts him more and more and makes his condition deplorable.

It is probable that most of the nervous currents travelling from different parts of our body to our brain never give rise to sensations. Our heart makes a noise every time it beats; our stomach churns the food with vigorous peristaltic movements. The wonder is that we are not in actual pain all the time. But these sensations are "subliminal." Let *c-d* (Fig. 117) be the limen, or threshold, of consciousness. We may suppose that only those sensations which are of sufficient intensity to rise above this line are noticed. That is, we should be unconscious of *g* and just feel *e*, but *f* would probably be a strong sensation, even a severe pain. The great majority of sensations, including particularly all from the

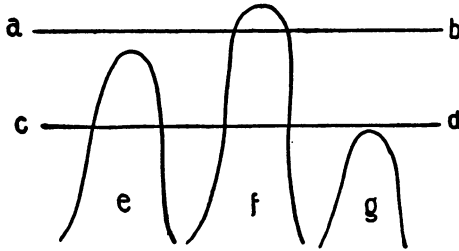


FIG. 117. A diagram of our sensations. *a-b*, the limen of consciousness when we are not paying strict attention to our sensations; *c-d*, the limen when we are paying strict attention to them. The sensation, *e*, will be perceived if the limen is *c-d*, and not noticed if it is *a-b*. The sensation, *f*, will be just perceived if the limen is *a-b*, and will be painful if it is *c-d*. The sensation, *g*, will escape notice even by the person whose limen is *c-d*. Most of the stimuli to the brain are like *g*.

natural operations of our organs, are subliminal. So we do not hear our heart beat, feel our stomach contract, etc. But this limen is not a constant point, for we are continually raising or lowering it. When we are intensely interested in a book, for instance, we do not hear our friend speak to us, or see near us something that under ordinary conditions would attract our notice. The limen for these sensations has been raised. Again, we are intensely interested and strain the eye to see, or the ear to hear, or the fingers to feel that which interests us. We lower the limen to these sensations, and stimuli which under ordinary conditions would be subliminal are perceived. This is practically what the neurasthenic has done. He has permanently lowered the limen for sensations from that organ which worries him, and so feels many more, and all of them more acutely, than does the normal man.

This man is sure he has heart disease, and his heart may actually pain him severely. This man has so severe sensations from his stomach that he rolls in agony on the floor. He suffers so much that he will consent to any operation, will even commit suicide; and yet the nervous currents which travel to his brain and give rise to these sensations need not be any stronger than those in some man who, because his limen is at a higher level, hardly notices the sensations from the stomach.

Let us change the figure. Look at this insect under the microscope. It is only a dot, and you cannot make out any details. Now turn down objective III. The field, or the circle of light which you see, is of exactly the same size, but the insect now almost fills it. Now turn down objective VI. The field is still of the same size, but only a part of the insect can be seen at one time. Try lastly the oil objective, and only a tip of one leg or wing can occupy this same field, so much has the insect become magnified. But again examine the specimen itself. The insect has not changed at all in size; we have merely changed the lenses through which we looked. So it is with sensations. The same stimuli may be transformed into sensations of so different magnitudes that they may be scarcely felt, or felt distinctly, or felt acutely, or felt with painful intensity; and yet the nervous current is the same for all; it is the "lens of the cortex" that is changed.

Neurasthenics are usually worried by the sensations from some one organ. This man hears of a disease and begins to study his sensations to see if he has any of its symptoms. The advertisements of patent medicines state that given symptoms indicate certain diseases. They are adapted to making the reader a neurasthenic with the symptoms that the medicine professes to cure. Sometimes a guilty conscience chooses the organ. But more often the organ chosen has been misused, and, although we can see no abnormality in it, we believe that it has reason for sending stimuli of unusual severity to the brain. The gastric neurasthenic, for instance, has generally misused his stomach for years by improper habits of eating. There is usually some little irregularity of action of the heart in the cardiac neurasthenic.

It may be asked why patients with organic disease and manifest reasons for pain are not all worse neurasthenics

than the men mentioned above. Some of them are so, but they are surprisingly few, and this fact emphasizes the importance of nervous fatigue, of a keen mind, and of an abundance of erroneous notions in the production of a first-class neurasthenic.

We have already mentioned the lack of relation between the seriousness of a disease and the severity of its symptoms. We add here that nature is kindly, and that with disease, especially one caused by germs, she usually sends an anæsthetic which soothes the pain. Very few persons suffer much while acutely ill, provided they are not subjected to superfluous ministrations; for the too solicitous care of a loving relative can add greatly to a patient's misery. This is the great surprise to hospital visitors, who expect disease and suffering to be correlated. They look down the rows of beds and ask, "Is any one here really ill?" Most persons gradually become dull, then stupid, then drowsy, then unconscious as death draws near. Some patients are partially anæsthetized, "toxic," during the whole of a fever. This makes typhoid fever a relatively "comfortable" disease for the patient, and confuses us much when we are trying to determine whether or not there is developing a peritonitis from perforation. Peritonitis due to appendicitis is a rather painful disease, but peritonitis due to perforation is not so painful. This patient cried out with pain. Fifteen minutes later, when the doctor came hurrying to the ward, the patient was asleep. When awakened, he was comfortable, but vexed that his slumbers were disturbed, and denied that he had had or then had any pain. He wanted to be let alone. He complained of almost no pain when the abdomen was then palpated, and yet even at that time a fatal peritonitis was developing. In fact surprisingly few very ill persons are really in much or any physical pain. Of course the case is quite different with the surgical patients and with patients who have any of the various colics—hepatic, renal, etc.—belonging really in the "traumatic" group.

But the neurasthenic suffers acutely. He gets angry when truthfully assured that the "terrible pain" around his heart does not denote heart disease, and that in real heart disease the heart itself seldom gives pain. He cannot understand why the "unbearable pains" in his back do not indicate

Bright's disease, although really serious Bright's disease is painless, so far as the kidneys are concerned. His pains are "terrible" and "unbearable," as some neurasthenics prove by their suicide, but for just that reason we doubt that he has organic disease, or even any disease at all. The severity of his pain makes us skeptical as to its seriousness. This is well illustrated almost daily at the entrance door of all large hospitals. "Doctor, come at once. The man is suffering terribly. He is screaming with pain," telephones the attendant, who admits new cases, to the doctor. And the doctor finishes what is then occupying him, muttering to himself, "Neurasthenic, probably." "New patient," says this same attendant on another occasion, "but he seems very comfortable;" and the doctor with a little experience hurries, for he knows that, while the chances are there is no need of haste, among those "comfortable" men are many very ill, but anæsthetized by the toxins of their disease.

Neurasthenia presupposes nervous bankruptcy. Nervous force is somewhat like money. We have a certain reserve capital of this force, and a certain daily income, and we daily expend a certain amount. Those whose income is large, or whose expenses are small, seldom suffer. But those whose income is small, or who are extravagant in their use of it, sooner or later come to nervous bankruptcy. Those who inherit a "nervous disposition," which usually means a small bequest of nervous force, and who eat poorly, sleep poorly, work hard, and, above all else, worry, are the nervous spend-thrifts. It does not signify how much a man works, or how important or pressing his work is; it does signify how much nervous force is being expended in doing it, and how much the man has. And any one can guess at the result. Some persons in performing a simple task spend twice as much nervous force as others in doing a much harder one.

Of all the "leaks" of nervous force, worry is the worst. Unfortunately, for nervous exhaustion, the body does not build an antidote, which could make it a "self-limiting disease," but, the weaker the neurasthenic gets, the more hopeless becomes his condition, for he sleeps progressively worse, or he eats less and less, or he worries about his condition more and more, all of which means a geometrical progression downward to nervous bankruptcy. Then begin the symptoms

of mental fatigue. He cannot apply himself to mental work; he is "too tired to think." If he is a business man, he may make bad errors in his business and thereby lose money; if he is a doctor, he may make errors in his practice and thereby endanger his patients. Patients suffering from mental fatigue are incapable of decision, anxious, "absent-minded," or they are egotistical, irritable, and extremely uncomfortable to deal with. They are very emotional. They have fears, or "phobias." (In this chapter we include the psychasthenics with neurasthenics.) Some will not cross an open square, some fear the dark, some cannot comfortably be alone, etc. Certain symptoms are very common: the headache on the top of the head; the pain at the back of the neck and at the lower end of the spine; muscular weakness; dizziness; a rapid, irregular heart; a painfully throbbing aorta; nausea; vomiting; diarrhoea; skin flushes and itching; and a host of other symptoms.

The diagnosis depends on the symptoms mentioned above and on our ability to exclude other diseases, which is difficult to do, as some grave diseases begin with marked symptoms of neurasthenia and very few signs of disease. This is true of Basedow's disease, tabes, dementia paralytica, and even of typhoid fever.

The treatment of neurasthenia deserves the most careful study. Each case must be treated individually, and yet there are several general rules which hold in practically all cases. The treatment should be rigid in every detail, else little may be gained. To carry it out 100% well means a cure; only 90% well gives almost as bad a failure as only 10% well. The proof of this is that one can accomplish as much (or more) in six weeks in a hospital as in six months at home. The object of the treatment is reconstruction and re-education—a *new* person. The patient is a nervous bankrupt. He must first allow his capital to reaccumulate, and then learn how in the future to live within his income of nervous force. He must be completely isolated from that environment in which his neurasthenia developed. He is safest in a room in a hospital which makes a specialty of such cases, and without a letter, newspaper, verbal message, or visit from the world outside. If the patient will not voluntarily agree to this, we refuse the case. A compromise of even 5% in the isolation is not to

be allowed. It is to be perfect, or not attempted at all. Of course each patient is "sure he cannot stand it," and each wife, husband, mother, or father is sure his or her visits will help and not hinder in the treatment. We do individualize patients in many respects, but, if we are sure the diagnosis "neurasthenia" is correct, in this respect almost never. It is interesting to observe how soon the mind is at rest, almost drowsy, and ready for recreation, after the patient really feels that every wire to the outer world is cut. If we find that he is not doing well, it is wise to watch and see whether he is not on the outlook for some familiar figure hovering around to catch a glimpse of him even from a distance, or coming to inquire about his progress. We wish him to attain repose, rest of mind and body, with the fewest possible reminders of the pleasures and the pains (for they always go hand in hand) of his previous life, in which he met his nervous downfall.

Since there is always fatigue—physical usually, mental often, nervous always—the patient should spend the first three or four weeks in bed. For some very sedentary persons who sleep well this period is too long, but it is usually necessary. It should be promised the patient that the more nearly horizontal he keeps his body, the less he sits up—even on one elbow—the more relaxed all his muscles are, the sooner he will be up. It is well to have the bed outdoors during the day and even during the night.

Insomnia is one of the important symptoms, and should not be combated, except for the first week perhaps, by drugs. The cold pack, the warm bath, and a cup of hot milk will help the patient sleep, but anxiety should not be felt, should much less be shown, if he doesn't sleep at all well. He will sleep well in time, and a little sleep goes a long way. It is well not to ask him how he slept and to discourage any worry on his part. As a rule, neurasthenic patients sleep much better than they think.

The diet is most important. These patients should be overfed. One does well to commence, as Dubois advises, with a week of pure milk diet unmixed with other foods, beginning with small amounts and gradually increasing to three, four, or even five quarts of milk a day, and then suddenly, unexpectedly to the patient, to change to a mixed diet of even

appalling abundance. The chances are that our patients have suffered from gastric symptoms. The chances are that their doctors have given various diets, one cutting out "acid foods," another starches, another fats, etc., and many cases come in a half-starved condition and afraid to eat almost everything. But if our diagnosis of neurasthenia is correct, the stomach, however much pain it may have caused, can and should digest painlessly almost anything within reason, and will do so if it must. The stomach is like a spoiled child. The more it is considered, petted, coddled, the more unruly it becomes; the more attention it demands, the more pain it gives. It needs figuratively to be whipped into subjection. After a few days of rebellion it usually handles well all the foods given, including those which previously caused discomfort, and after that all generally goes well. It is interesting that one should need to prescribe food. The patient is told that everything on the tray is medicine, to be eaten to the last crumb or drunk to the last drop. Some patients who suffer much from a certain dish as food will not suffer at all from the same dish eaten as medicine.

Of course, if the patient insists that a certain vegetable has always distressed him, it is well to "forget" to serve that, but he should not for an instant think that the omission is a concession to his tastes, for no small point is gained by a demonstration that there is nothing which he cannot eat without discomfort. He should gain weight. The gain is in itself unimportant, although it usually is encouraging, but nervous force seems to be stored up best during periods of gain in flesh.

Hydrotherapy is very valuable. The cold pack at night and the cold sponge bath in the morning are among the best tonics of the nervous system, but some patients sleep better after a warm bath in the evening.

Many patients are constipated, and this should be overcome through habit and diet. The patient should attempt to have a movement every day at just the same time. The diet should be rich in foods containing much fibrous tissue, such as fruits and green vegetables. A recent patient who had regularly taken a laxative for twelve years was able in three weeks to form a habit so fixed that often "he did not need to look at his watch to see when nine o'clock came."

After resting in bed for the prescribed time the patient uses a back-rest, then sits up in a chair, then walks. Then begins the most important part of the cure, for all the symptoms on account of which he came to the hospital return with increased violence. His whole day must be full; there must be something for each hour,—reading, walking, resting, and working. The amount of exercise should be increased daily, until, if possible, his best previous records have been broken. Now is the time to re-educate him, to show him that the things which he thought he could not do he now can do. Tell him that he was like an electric automobile run down. Its recharging is a slow process, and while it is going on there is little to show, but the force is evident when the machine is put to work. Then comes a transitional vacation away from work and from hospital atmosphere, finally a return to home and work. The patient should have the strength, energy, and enthusiasm to return to work, and the recently acquired rules and habits so ingrained that he will not relapse.

Hysteria.—Hysteria may be defined as a disease in which the mind produces objective signs as well as subjective symptoms of organic diseases. It may be described as the unconscious and skilful imitation of almost any disease, especially a serious one, with its signs and symptoms almost exactly reproduced, and with certain additional signs peculiar to hysteria and called the “stigmata” of this disease. While it is most common in women, it occurs in men also, and it spares no age, the form present in children under ten being one of the hardest to detect.

The first broad division of this disease separates the convulsive from the non-convulsive forms, of each of which we give a few illustrations. The minor convulsive form may be illustrated by the attacks of alternate laughing and crying which the lay mind associates with “hysteria.” Other patients have convulsions which may resemble epilepsy so closely that only an expert can tell the differences. One of the differences which it is most important to observe is that during the convulsion the patient may hurt others but never hurts himself. He never falls “hard,” as if shot, and he never chews his tongue. The major convulsive type is seldom seen in America. Patients with this form have a series of convulsive attacks which lasts for several days.

Among the non-convulsive forms is the common hysterical paralysis. There is no type or form of organic paralysis which may not be simulated in hysteria (Osler); hemiplegia, paraplegia, monoplegia, all occur. Of course, between these and the paralyses due to organic disease there are differences, of which the most important is that the hysterical paralysis suddenly gets well. But the muscle is as truly paralyzed in the hysterical as in the organic type,—that is, the will is unable to contract it.

Many of the incurable diseases of the spinal cord—lateral sclerosis, insular sclerosis, etc.—may be mimicked so successfully that the later recovery alone shows us the error in diagnosis. Various forms of rhythmical spasm, of tremors, etc., may continue for months. Parts or one half of the body may become perfectly anæsthetic to touch, pain, etc., or, on the contrary, may be exquisitely hypersensitive. Some hysterical patients are blind, some are deaf, for months. Among the hysterical pains, which are very severe, are the following: backache; pains, in the gastric region, simulating the pains of gastric ulcer; pains, in the appendix, simulating the pains of appendicitis; especially, pains in the pelvis. These gastrointestinal features may be very distressing. The extreme pain, nausea, vomiting, including the true fecal vomiting (supposed to be due only to intestinal obstruction), even hemorrhage from the stomach, the extreme constipation or violent diarrhoea, and not only loss of appetite, but aversion to food, may lead to the most extreme emaciation and death.

Some hysterical patients have a very rapid pulse, some a very slow one. Some have dyspnoea, some a hiccough which may last months. Some have spasm of the respiratory muscles which causes peculiar cries, whoops, or noises mimicking animals.

There are cases of hysterical rheumatism in which the joints are acutely inflamed,—that is, become swollen, red, hot, painful when touched or moved. And, finally, there is an hysterical fever, which may run a course like meningitis, with stiff neck; or like peritonitis, with abdominal pain; or like consumption, with bloody sputum.

The diagnosis of hysteria depends on the past history of the patient, on the slight variations between the case and the disease which it simulates, but especially on the discovery

of the stigmata of hysteria. These include the following: sighing respiration—that is, a “catching of the breath” every fifth or sixth respiration; the very common “globus hystericus”—the sensation of a ball rising in the throat; the areas of anæsthesia, usually between the nipples; the contracted fields of vision; the tendency to emotional outbreaks; and convulsive seizures of various kinds.

In judging of these cases one is apt to be far too unkind. There often is a tendency to deception, but, for the most part, these patients believe in their diseases more firmly than do their friends. They are usually “unfortunate in their parents” and inherit strong neuropathic tendencies; they have usually been even more unfortunate in their early training and have never learned to control themselves; and they are often so unfortunate as to employ doctors who treat seriously their various ailments.

The treatment is that for neurasthenia, vigorously carried out, especially so far as isolation is concerned, and with the emphasis laid on the re-education. Here the trained nurse who is also a specialist is not only important, but indispensable. The patient must be handled with the greatest tact and firmness, and taught how to control himself.

Many specialists believe that the condition is congenital and should be dealt with chiefly in closed institutions.

INDEX

- Absoess, of intestines, 127
 - of liver, 140, 327
 - of pancreas, 145
- Acid intoxication, 224
- Acute exanthemata, 307
 - lobar pneumonia, 249
 - nephritis, 168
 - pancreatitis, 145
 - rheumatic fever, 302
 - tuberculous pneumonia, 256
- Addison's disease, 219, 280
- Adenoids, 75
- Adhesive pericarditis, 279
- Adiposis dolorosa, 217
- Adrenal glands, diseases of, 219
 - tuberculosis of, 280
- Adrenalin, 281
- Adventitia, 9, 39
- Aërophagia, 105
- Aestivo-autumnal malaria, 329, 330
- Air hunger, 224
 - passages, 70
 - swallowing, 105
- Albumin in urine, 152
- Albuminuria, 152
- Alimentary canal, diseases of, 85
- Ambulatory typhoid, 244
- Amœba coli, 326
- Amœbic abscess of liver, 141
 - dysentery, 326
- Ampulla of Vater, 135
- Amylopsin, 109
- Anacidity, 106
- Anæmia, 26
 - hæmorrhagic, 28
 - pernicious, 31
 - of poor, 29
 - of poor Whites, 335
 - primary, 30
 - secondary, 28
 - toxic, 29
- Anastomosis of vessels, 41
- Aneurism, 44, 47
- Angioneurotic cedema, 215
- Animal parasites, 325
- Ankylostoma duodenale, 335
- Ankylostomiasis, 335
- Ankylosis, 305
- Anopheles mosquitoes, 330
- Anterior poliomyelitis, 198
- Anthrax, 295
- Antiperistalsis, 120
- Antitoxin, 245
 - diphtheria, 289
- Antrum, 70
- Anuria, 158
- Aortic area, 54
 - insufficiency, 57
 - stenosis, 56
- Apathy, 352
- Apex beat, 54
- Aphasia, 188
- Apnoea, 346
- Apoplexy, 186, 206
- Appendicitis, 123
- Appendix abscess, 127
 - vermiformis, 108, 123
- Arachnoid, 282
- Arsenic therapy, 31
- Arteriosclerosis, 44
- Artery, 9, 11
 - of cerebral hemorrhage, 207
 - end, 41
- Arthritis, infectious, 304
 - rheumatoid, 305
 - secondary, 304
- Ascaris lumbricoides, 336
- Ascending colon, 108
- Ascites, 62, 138
- Asiatic cholera, 293
- Asthma, 80
 - cardiac, 346
- Ataxia, 202
 - locomotor, 297
- Atoxyl, 32
- Atrophic cirrhosis of liver, 136
 - nasal catarrh, 74
- Atrophy, progressive muscular, 206
- Auditory nerve, 193
- Aura of epilepsy, 210
- Autumnal fever, 73

- Bacillary dysentery, 290
 Bacilli, 236
 Bacilluria, 237
 Bacillus anthracis, 295
 coli communis, 112
 diphtheria, 286
 dysenteriae, 290
 influenzae, 284
 lactis aërogenes, 112
 leprae, 291
 mallei, 292
 paracolon, the, 236, 239
 paratyphoid, the, 236, 239
 pestis bubonica, 294
 tetani, 292
 tuberculosis, 256, 263
 typhosus, 236
 Bacteria, 231, 235
 nitrifying, 229
 Bacteriolysis, 246
 Basedow's disease, 218
 Beef tapeworm, 337
 Bell's palsy, 193
 Beriberi, 206, 323
 Bigeminal pulse, 347
 Bile, 130
 acids, 131
 ducts, 132
 pigment, 20
 Bilious fever, 244
 Biliousness, 116, 131
 Black death, 295
 measles, 311
 smallpox, 314
 plague, 294
 vomits, 321
 -water fever, 331
 Bleeders, 34
 Blood, 11
 -building organs, 15
 -cells, 11
 red, 17
 white, 20
 -clot, 11
 coagulation, 11
 -corpuscles, 11
 count, 27
 crisis, 28
 diseases, 26
 faking, 18
 -plasma, 11, 15
 -platelets, 26
 poisons, 51
 -serum, 11
 stroma, 18
 -vessels, 38
 Blue baby, 62
 Body heat, 14
 Boil, history of, 21
 Bone-marrow, 19
 Bone, tuberculosis of, 280
 Bothriocephalus latus, 337
 Bowel, infarct of, 122
 Bowman's capsule, 155
 Bradycardia, 350
 Brain, softening of, 207
 tumor of, 190
 Break-bone fever, 322
 Break in compensation, 57
 Brick-dust sediment, 165
 Brick makers' anaemia, 335
 Bright's disease, 153
 Broca's convolution, 187
 Bronchiectasis, 83
 Bronchitis, 76
 acute, 79
 chronic, 80
 fibrinous, 80
 putrid, 80
 Bronchopneumonia, 79
 Bubonic plague, 294
 Calcium, 34
 Calculi, pancreatic, 146
 renal, 181
 Calyces, renal, 157
 Camp fever, 322
 Cancer of liver, 143
 of oesophagus, 85
 of stomach, 101
 Capillaries, 11, 39, 40
 Capillary pulse, 350
 Caput Medusae, 139
 Carbohydrates in urine, 152
 Carbon monoxide hæmoglobin, 67
 Cardalgia, 94
 Cardiac asthma, 80, 346
 compensation, 57
 orifice of stomach, 89
 spasms, 105
 Cardio-vascular system, 40
 Carnivorous animals, 149
 Casts in urine, 153
 Catarrh, 73
 Catarrhal jaundice, 133
 Cathartics, 117
 Cavity formation, 256
 Cell, 7
 blood-, 11
 red, 17
 white, 11, 20
 Cerebellum, 183, 189
 Cerebral embolus, 43, 207
 hemorrhage, 206

- Cerebral hemorrhage, artery of, 206
 Cerebrospinal meningitis, 282
 Cerebrum, 183
 Chalk-stones, 221
 Chancre, 296
 Chemotaxis, 23
 Cheyne-Stokes respiration, 346
 Chicken-pox, 315, 316
 Chilblains, 215
 Child crowing, 78
 Chlorosis, 30
 Cholangitis, 140
 Cholecystitis, 133
 Cholera, 293
 Cholesterin, 131
 Chords tendinæ, 37
 Chorea, 213
 chronic, 214
 insaniens, 213
 Chronic interstitial nephritis, 173
 pancreatitis, 146
 parenchymatous nephritis, 170
 passive congestion, 61, 137
 pulmonary tuberculosis, 259
 rheumatism, 305
 Chyme, 90
 Ciliata, 328
 Circulation of blood, 34
 greater, or systemic, 53
 lesser, or pulmonary, 53
 Cirrhosis of kidney, 173
 of liver, 136
 Claw hand, 206
 Clot, blood, 11
 Club foot, 199
 Coagulation of blood, 11
 Coarsely granular leucocytes, 24
 myelocytes, 25
 Cocci, 235
 Coccydynia, 192
 Cold abscess, 280
 on chest, 79
 in head, 72
 Colic, gall-stone, 135
 Collateral circulation, 42
 Colon, 108
 Color index, 27
 Coma, 352
 diabetic, 224
 Common duct, 132
 Compensation, cardiac, 57
 Confluent smallpox, 313
 Congenital heart disease, 62
 Congestion, active, 62
 chronic passive, 61, 137
 Conglomerate tubercles, 258
 Constipation, 144
 Constitutional diseases, 220
 Consumption, 256
 of bowels, 270
 Continuous fever, 244
 Contractures, 306
 Convoluted tubules, 153
 Convulsions of brain, 184
 Convulsions, 210
 Cord, spinal, 194
 Coronary arteries, 43
 Corpuscles, blood, 11
 Cortex of brain, 183
 of kidney, 157
 Coryza, 72
 Cough, morning, 80
 winter, 80
 Cranial nerves, 190
 Creatinin, 151
 Creeping pneumonia, 252
 Creosote inhalations, 84
 Cretin, 217
 Crisis, blood, 28
 pneumonia, 252
 temperature, 344
 Cross-legged progression, 200
 Croup, membranous, 287, 346
 spasmodic, 78
 Crowing, child, 78
 Crusts, skin, 313
 Culex mosquitoes, 331
 Cylindrical bronchiectasis, 84
 Cystic duct, 132
 kidney, 182
 Cysticercus disease, 338
 Dandy fever, 322
 Delirium cordis, 347
 Dementia, paralytica, 194
 Dengue, 322
 Dermum's disease, 217
 Dermatitis, acute exfoliative, 309
 Descending colon, 108
 Diabetes insipidus, 161, 226
 mellitus, 14, 147, 220, 222
 Diabetic coma, 224
 Diarrhoea, 112
 fatty, 147
 pancreatic, 147
 Diastase, 109
 Diastole, 36, 54
 Dicrotic pulse, 348
 wave, 55
 Dietl's crises, 178
 Diffuse bronchiectasis, 84
 Diffusion of gases, 66
 Digestion, 85
 Dilatation of heart, 63

- Dilatation of stomach, 95
 Diphtheria, 206, 286, 309
 Diplegia, 200
 Diplococci, 235
 Diplococcus intracellularis meningitidis, 282
 Diplococcus lanceolatus, 249
 Dissecting aneurism, 47
 Diuretics, 161
 Double pneumonia, 252
 Dropey, 14, 147
 Dumb chills, 330
 Dum dum fever, 333
 Duodenum, 108
 Dura mater, 282
 Dysentery, 114
 Dyspepsia, 103, 290
 Dyspnea, 345

 Echinococcus disease, 340
 Egyptian chlorosis, 335
 Elephantiasis, 334
 Embolism, 42
 cerebral, 207
 Embolus, 42
 Emphysema, 82
 End artery, 41
 Endocarditis, 49
 acute, 50
 chronic, 53
 simple, 50
 ulcerative, 50
 Enterokinase, 109
 Enteroptosis, 96, 122
 Eosinophile cells, 24
 Eosinophilia, 25
 Epidemic parotitis, 317
 Epilepsy, 210
 Jacksonian, 185
 Epileptiform fits, 212
 Epinephrin, 281
 Epistaxis, 71
 Erypsin, 111
 Erysipelas, 298
 Erythema, 307
 Erythromelalgia, 215
 Ethmoid sinuses, 70
 Eustachian tubes, 75
 Exanthemata, 307
 Excrements, 150
 Excretion, 150
 Exfoliative dermatitis, 309
 Exophthalmic goitre, 218
 Exophthalmos, 218
 External respiration, 66

 Facial neuralgia, 191
 paralysis, 193
 Famine fever, 298
 Farcy, 292
 Fatty casts, 153
 diarrhoea, 147
 heart, 63
 necrosis, 145
 Fecal vomiting, 120
 Fibrin, 11
 Fibrinous bronchitis, 80
 Filaria Bancroftii, 333
 Filariasis, 333
 Finely granular leucocytes, 20
 myelocytes, 25
 "Fish eyes," 336
 "Fits," 210
 Flagellata, 328
 Flail-joints, 199
 Floating kidney, 177
 "Florida complexion," 336
 Focus of infection, 234
 Follicles, solitary, 238
 Follicular tonsillitis, 300
 Foods, 12, 87, 148
 Food-stuffs, 87, 148
 Foot drop, 205
 Frontal sinuses, 70
 Function of blood, 12
 of cells, 10
 Functional albuminuria, 153
 Fusiform aneurism, 47

 Gall-bladder, 132
 -ducts, 132
 -stone, 133
 colic, 135
 Galloping consumption, 262
 Ganglia, 190
 Gastralgia, 107
 Gastric crises, 103
 juice, 90
 Gastritis, acute, 92
 chronic, 93
 Gastroptosis, 96
 General paralysis, 194, 297
 German measles, 312
 Germicidal bodies, 23
 Germs, 231
 Glanders, 292
 Glands, diseases of, 216
 Gleet, 298
 Globus hystericus, 372
 Glomerulus, 155
 Glosso-pharyngeal nerve, 193
 Glottis, cedema of, 78
 Glucose, 16, 87, 130, 152

- Glycogen, 130
- Glycosuria, 222
- Goitre, 217
- Gonococcus, 298
- Gonorrhoea, 298
- Gout, 220
- Grand mal, 210
- Graves's disease, 218
- Greater curvature of stomach, 90
- Grippe, 284
- Ground itch, 336
- Gummata, 297
- Habit spasm, 214
- Hæmoglobin, 17
 - carbon-monoxide, 67
- Hæmophilia, 34
- Hemorrhage, anemia of, 11, 28
 - cerebral, 206
 - pulmonary, 261
 - tuberculosis, 268
 - typhoid fever, 241
- Hemorrhagic pancreatitis, 145
 - smallpox, 314
- Harrison's groove, 76
- Hay fever, 72
- Headache, 214
- Hearing, nerve of, 193
- Heart, 34
 - burn, 94
 - dilated, 63
 - diseases of, 49, 63
 - sounds of, 54
- Heat, body, 14
 - regulation, 341
- Hematogenous jaundice, 132
- Hematuria, 154
- Hemiorania, 214
- Hemiplegia, 186, 200, 208
- Hemorrhoids, 139
- Hepatic duct, 132
 - fever, 136
- Hepatization, 249
- Hepatogenous jaundice, 132
- Herbivorous animals, 149
- Hernia, 119
- Herpes zoster, 204
- Hobnail liver, 137
- Hookworm disease, 335
- Horse-shoe kidney, 157
- Hour-glass stomach, 90
- Hospital fever, 322
- Hunch-back, 280
- Huntington's chorea, 214
- Hyalines, 329
- Hydrocephalus, 183
- Hydrochloric acid, 90
- Hydrolysis, 110
- Hydromyelia, 202
- Hydronephrosis, 176
- Hydrophobia, 323
- Hydrothorax, 62, 170
- Hyperacidity, 106
- Hyperpyrexia, 343
- Hypersecretion, 106
- Hyperthyroidism, 217
- Hypertrophic cirrhosis, 139
- Hypertrophy, 45
 - of turbinate bones, 73
- Hypoglossal nerve, 194
- Hypothermia, 241
- Hysteria, 370
- Hysterical epilepsy, 212
- Ileo-cæcal valve, 108
- Ileum, 108
- Immunity bodies, 15
- Impaction, fecal, 115
- Incubation, 235
- Infarction of bowel, 122
- Infarcts, 42
- Infection, 233, 234
 - secondary, 234
 - terminal, 234
- Infectious arthritis, 304
 - diseases, 229
- Inflammation, 22, 233
 - of bowels, 127
- Influenza, 284
- Insane, general paralysis of, 194
- Insomnia, 368
- Insufficiency, aortic, 57
 - mitral, 59
 - tricuspid, 61
 - valvular, 50
- Intercostal neuralgia, 192
- Intercurrent relapse, 240
- Intermittent fever, 344
- Internal capsule, 43, 185
- Internal respiration, 65
- Interstitial nephritis, 173
- Intestinal fluid, 109
- Intestine, diseases of, 108
 - infarct of, 122
 - obstruction of, 118
 - ulcers of, 114
- Intima, 9, 38
- Intussusception, 119
- Invertase, 111
- Invertin, 109
- Involuntary muscle, 38
- Iodide of potassium, 47

- Jacksonian epilepsy, 185, 212
 Jail fever, 322
 Jaundice, 132
 Jejunum, 108
 Joints, tuberculosis of, 280
 June cold, 73

 "Kernels," 270
 Kidneys, 154
 diseases of, 148, 166
 tuberculosis of, 280
 Knots in bowel, 120
 Koch's laws, 231
 Koplik's spots, 310

 Lactase, 109
 Lacunæ, 206
 Laking of blood, 18
 Large mononuclear leucocytes, 24
 Larval pneumonia, 252
 Laryngismus stridulus, 78
 Laryngitis, 76, 78
 Larynx, oedema of, 78
 Leishman-Donovan bodies, 333
 Leprosy, 291
 Lesser circulation, 53
 Lesser curvature of stomach, 90
 Leucocytes, 11, 20
 Leucocytosis, 25
 Leucopenia, 25
 Leukemia, 32
 Lientery, 113
 Lightning pains, 203
 Lipase, 109
 Limit, carbohydrate, 222
 Liver, abscess of, 140, 327
 cancer, 143
 diseases of, 129
 Lobar pneumonia, 249
 Lobular pneumonia, 251
 Lobule of liver, 129
 Local lesion, 234
 Lockjaw, 292
 Locomotor ataxia, 202
 Low fever, 244
 Lues, 296
 Lumbago, 192, 306
 Lumbar puncture, 284
 Lungs, 66
 diseases of, 82
 Lymph, 14
 Lymphatic glands and vessels, 40
 leukemia, 33
 Lymphatism, 301
 Lymph-glands, tuberculosis of, 266
 Lymphocytes, 24
 Lysis, 252, 344

 Macule, 312
 Malaria, 328
 organisms of, 328
 pernicious, 328
 Malarial typhoid fever, 244
 Malignant diphtheria, 308
 pustule, 296
 Malta fever, 299
 Maltase, 111
 Maximum cardiac impulse, 54
 Measles, 312
 Measles (of meat), 338
 Media, 9, 38
 Mediterranean fever, 299
 Medulla, 183
 Medusa, head of, 139
 Megaloblasts, 31
 Membranous croup, 287, 346
 Meninges, 183
 Meningitis, 272, 282
 Meniscus, 162
 Metabolism, diseases of, 220
 Metastases of cancer, 101
 Micrococcus gonorrhoeæ, 298
 lanceolatus, 249, 255
 melitensis, 299
 Migraine, 214
 Miliary tuberculosis, 271
 Miners' anemia, 335
 Mitral area, 54
 insufficiency, 59
 stenosis, 60
 Mononuclear leucocytes, 24
 Morning cough, 80
 Mosquitoes, Anopheles, 330
 Culex, 331
 Stegomyia, 320
 Motor area of cortex, 43, 184
 aphasia, 188
 nerves, 190
 neurons, 198
 neuroses, gastric, 104
 • Moulds, 232
 Mouth breathing, 76
 Mucous colitis, 122
 Multiple neuritis, 203, 205
 Mumps, 317
 Mural thrombi, 49
 Muscular atrophy, 206
 rheumatism, 306
 Myelocytes, 25
 Myelogenous leukemia, 32
 Myocarditis, 62
 Myxœdema, 14, 216

- Nasal spur, 73
 Nasopharynx, 70, 75
 Neapolitan fever, 299
 Necrosis, fatty, 145
 of pancreas, 145
 Nephritis, 153, 167
 Nephrolithiasis, 180
 Nerves, 190
 cranial, 190
 spinal, 196
 Nervous diarrhoea, 113
 dyspepsia, 103
 fever, 244
 system, 183
 vomiting, 105
 Neuralgia, 190
 facial, 191
 red, 215
 Neurasthenia, 362
 Neuritis, 203
 multiple, 205
 Neurons, 198
 Neuroses, gastric, 103
 Neutrophile cells, 23
 Night cries, 76
 Nitrifying bacteria, 229
 Nitrogenous food, 149
 Nitrogen in urine, 151
 Nitroglycerine, 47
 Non-nitrogenous foods, 149
 Normoblasts, 19
 Nose, 70
 Nosebleed, 71
 Nostrils, 70
 Nubecula, 165
 Nucleated red cells, 19
 Nucleus, 7
 Nutrition, disturbances of, 220

 Obliterative appendicitis, 124
 Obstruction, intestinal, 118
 nasal, 76
 (Edema, angioneurotic, 215
 of glottis, 78
 of larynx, 78
 (Esophagus, diseases of, 85
 varices of, 138
 Olfactory nerves, 190
 Oligopnoea, 345
 Optic nerves, 189, 190
 tract, 189
 Organ, 7
 Orthopnoea, 171, 345
 Orthostatic albuminuria, 152
 Osmosis, 66
 Oxybutyric acid, 224

 Oxyhæmoglobin, 18, 67
 Oxyuris vermicularis, 337
 Ozæna, 74

 Pain as a symptom, 356
 localisation, 360
 projection, 360
 Palsy, 209
 Papules, 312
 Pancreas, 144
 Pancreatic abscess, 145
 diarrhoea, 147
 juice, 111
 secretion, 14, 111
 Pancreatitis acute, 145
 chronic interstitial, 146
 hemorrhagic, 145
 gangrenous, 145
 Pantomime fever, 322
 Paracolon bacilli, 236, 239
 Paralysis, 43
 of adults, 200
 agitans, 209
 anterior poliomyelitis, 198
 facial, 193
 general, of insane, 194
 muscle, 198
 spastic, 200
 stroke of, 206
 Paraplegia, 200
 spastic, 201
 Parasites, animal, 325
 Parathyroids, 219
 Paratyphoid organisms, 236, 239
 Parenchymatous nephritis, 170
 Paresis, 194
 Parkinson's disease, 209
 Parotitis, 318
 epidemic, 31
 Paroxysmal tachycardia, 349
 Passion fits, 78
 Pelvis, renal, 157
 Pepsin, 90, 110
 Perforation in typhoid fever, 241
 Pericarditis, 171
 Pericardium, tuberculosis of, 277
 Pernicious anæmia, 31
 malaria, 331
 Peristalsis, 90
 Peristaltic unrest, 104
 Peritoneum, tuberculosis of, 279
 Peritonitis, 127
 Petechia, 322
 Petit mal, 210
 Peyer's patches, 238
 Phagocytosis, 21, 23
 Phobias, 367

- Phosphates, 152
 sediment of, 165
 Photophobia, 284
 Pia mater, 282
 Pigeon breast, 76
 Piles, 139
 Pinworm, 337
 Pits, 313
 Plague, 294
 Plasma, blood, 11, 15
 Plasmodium of malaria, 328
 Platelets, 11, 26
 Pleura, tuberculosis of, 275
 Pleurisy, 275
 Pleurodynia, 306
 Pneumonia, 249
 central, 253
 creeping, 252
 ether, 253
 larval, 252
 lobar, 249
 lobular, 255
 terminal, 253
 traumatic, 253
 Pneumothorax, 273
 Point of maximum impulse, 54
 Poisons, blood, 51
 Poker back, 305
 Poliomyelitis, 198
 Polycythemia, 27
 Polymorphonuclears, 23
 Polypnea, 345
 Polyserositis, 274
 Polyuria, 159, 226
 Pons, 183, 190
 Pork worm, 337
 Portal circulation, 36
 obstruction, 137
 Portal of entry, 51, 234
 Potassium iodide, 47
 Pot belly, 227
 Pott's disease, 280
 Preservation of urine, 166
 Progressive muscular atrophy, 206
 Protective bodies, 14
 Pruritus, 224
 Protoplasm, 7
 Protozoa, 325
 Pseudocrisis, 252
 Pseudopod, 21
 Ptyalin, 88
 Pulmonary tuberculosis, 256
 valve disease, 62
 Pulmonic area, 54
 Pulse, 346
 capillary, 350
 rate, 348
 Pulse, regularity, 347
 tension, 350
 wave, 37, 55
 Pulsus paradoxus, 279
 Pus, 22, 231
 Pustules, 313
 Putrid bronchitis, 80
 Pyemia, 234
 Pyelitis, 178
 Pylephlebitis, 140
 Pyloric spasm, 105
 Pylorus, 89, 90
 Pyogenic organisms, 231
 Pyonephrosis, 177, 178
 Pyramids, renal, 157
 Pyramidal tract, 185, 196
 Pyroplasmosis, 333
 Pyuria, 154

 Quartan malaria, 329, 330
 Quinsy, 301
 Quotidian fever, 330

 Rabies, 323
 Rag picker's disease, 296
 Raspberry tongue, 307
 Raynaud's disease, 215
 Reaction of urine, 164
 Rectum, 108
 Red blood-cells, 11, 17
 neuralgia, 215
 Referred pains, 360
 Reflex convulsions, 212
 Relapse, febrile, 240
 Relapsing fever, 298
 Remittant fever, 344
 Renal asthma, 80
 calculus, 180
 colic, 181
 tumors, 182
 Rennet, 90
 Reserve cardiac force, 56
 Respiration, external, 66
 internal, 65
 mechanism, 68
 movements, 68
 Respirations, 18, 345
 Respiratory centre, 346
 organs, 65
 Rheumatism, acute, 302
 chronic, 305
 muscular, 306
 Rheumatoid arthritis, 305
 Rickets, 227
 Rock fever, 299
 Romberg's sign, 203
 Rosary, rickety, 227

- Rose cold, 73
 spots, 240
 Round worm, 336
 Rumination, 105
 Rupture, 119

 Saber legs, 227
 Saccular aneurism, 47
 bronchiectasis, 84
 Saint Vitus's dance, 213
 Salivary digestion, 88
 Salivation, 297
 Salts in urine, 151
 Scarlet fever, 307
 Sciatica, 192, 204
 Scrofula, 260, 270
 Seatworm, 337
 Secretin, 110
 Secretion, 13
 external, 13
 internal, 13
 of gastric juice, 91
 Secretory cells, 156
 neuroses, 106
 Sediments in urine, 165
 Segments, 329
 Sensations, 358
 Sensorium, 358
 Sensory aphasia, 188
 nerves, 190
 neuroses, gastric, 107
 Septicæmia, 233
 Septum of nose, 70
 Serous sacs, tuberculosis of, 274
 Serum, blood, 11
 Seventh nerve, 193
 Shaking palsy, 209
 Shiga's bacillus, 290
 Ship fever, 322
 Shingles, 204
 Shock, 206
 Shooting pains, 203
 Sick headache, 214
 Sigmoid flexure, 108
 Signs of disease, 352
 Sinus disease, 74
 Sinuses of nose, 70
 Sleeping sickness, 333, 335
 Small mononuclear leucocytes, 24
 Smallpox, 312
 Softening of the brain, 207
 Solitary follicles, 238
 tubercles, 280
 Somnolence, 352
 Southern anæmia, 335
 Spasmodic croup, 78
 Spastic paralysis, 200

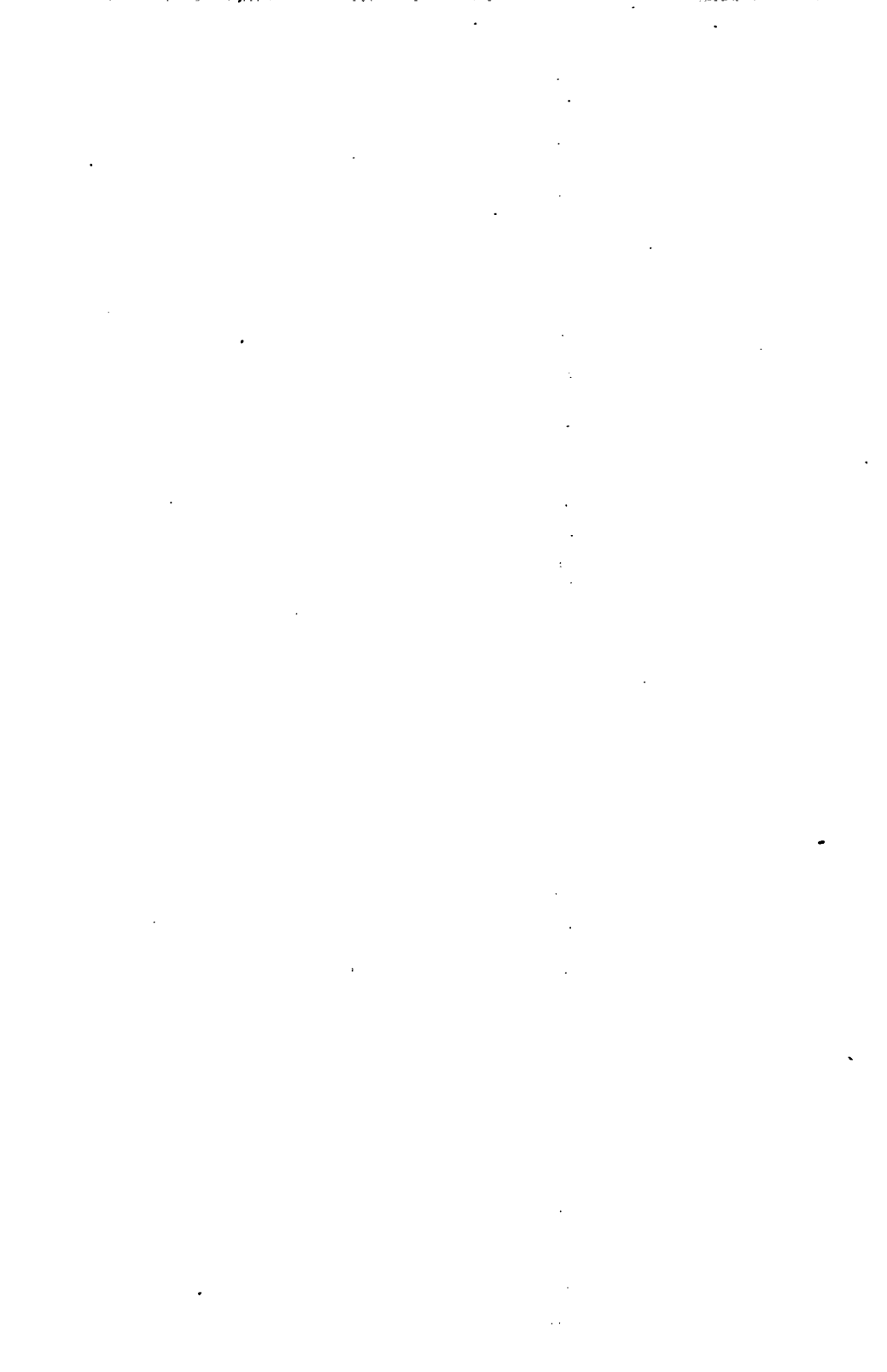
 Specific germs, 231
 gravity, 161
 toxins, 232
 Sphenoidal sinuses, 70
 Spinal accessory nerve, 194
 cord, 194
 nerves, 196
 Spirilla, 236
 Spirillum of cholera, 293
 Spirochæte Obermeieri, 298
 pallidum, 296
 Splenomegaly, tropical, 333
 Spotted fever, 282, 322
 Spur, 73
 Squamous epithelium, 77
 Staphylococci, 236
 Status lymphaticus, 301
 Steapsin, 109
 Stegomyia fasciata, 320
 Stenosis, 51, 55
 aortic, 56
 mitral, 60
 Steppage gait, 205
 Stiff neck, 306
 Stokes-Adams syndrome, 349
 Stomach, 89
 cancer of, 101
 dilatation of, 95
 diseases of, 85
 neuroses of, 103
 ulcer of, 97
 Stone, gall-, 133
 pancreatic, 146
 renal, 180
 Strangulated hernia, 119
 Strawberry tongue, 307
 Streptococci, 236
 Streptococcus pyogenes, 298, 300
 Stricture of œsophagus, 85
 Stroke, paralysis, 185, 206
 Stroma, 18
 Stupor, 352
 Subacute nephritis, 170
 Subliminal sensations, 363
 Subnormal temperature, 343
 Subsultus tendinum, 240
 Sugar in urine, 152
 Sunstroke, 344
 Superacidity, 106
 Supermotility, 104
 Suppression of urine, 158
 Suppurative tonsillitis, 301
 Symptoms of disease, 352
 Syphilis, 296
 Systole, 36, 54
 Systoles, extra, 348

- Tabes dorsalis, 202, 297
 mesenterica, 270
 Tachycardia, 218, 349
 Tænia echinococcus, 340
 Tapeworms, 337
 Temperature, 341
 Tenesmus, 114
 Tertian malaria, 328
 Tetanus, 292
 Tetany, 219
 Thoracic duct, 40
 Threadworm, 337
 Three day fever, 322
 Thrombosis, 41
 Thrombus, 41
 cerebral, 207
 mural, 49
 Thrush, 232
 Thyroid gland, secretion of, 14
 diseases of, 216
 extract, 216
 Tic, 214
 douloureux, 191
 Tissue, 8
 lymph, 14
 Tolerance to sugar, 222
 Tonsillitis, 300
 Tonsils, 75, 300
 Tophi, 220
 Torticollis, 194
 Toxins, 232
 Tracheitis, 76
 Tract, motor, 201
 pyramidal, 196
 sensory, 201
 Transverse colon, 108
 Trichina spiralis, 334
 Trichiniasis, 334
 Tricuspid area, 54
 insufficiency, 61
 Trigeminal pulse, 348
 Tropical splenomegaly, 333
 Trypanosoma gambiense, 333
 Trypanosomiasis, 333
 Trypsin, 108, 110, 145
 Trypsinogen, 108, 145
 Tubercle, 257
 bacillus, 256
 Tuberculin, 281
 Tuberculosis, 256
 of adrenals, 280
 of bones, 280
 of joints, 280
 of kidneys, 280
 of lymph-glands, 269
 of meninges, 272
 miliary, 271
 Tuberculosis of pericardium, 277
 of peritoneum, 279
 of pleura, 275
 of serous membranes, 273
 Tuberculous toxæmia, 271
 Tubules, renal, 155
 Tumors, brain, 182
 kidney, 182
 (see cancer)
 Tunnel digger's anæmia, 335
 Turbinate bones, 70
 Twilight condition, 352
 Twists of bowel, 120
 Typhoid bacillus, 236
 fever, 236
 Typhoidal fever, 244
 Typho-malaria, 244
 Typhus fever, 322

 Ulcerative endocarditis, 50
 Ulcers of intestine, 114, 238
 of stomach, 97
 Uncinaria americana, 335
 Uncinariasis, 335
 Uræmia, 151, 166
 Urate sediment, 165
 Urea, 16, 130, 151
 frost, 150
 Ureter, 157
 Urethritis, 298
 Uric acid, 151
 Urine, 150
 Urinary gravel, 165
 Uric acid, 151
 Urinometer, 162

 Vaccination, 315
 Vaccine, 315
 Vaccinia, 315
 Valvular heart disease, 53
 insufficiency, 50, 55
 stenosis, 51, 55
 Varicella, 316
 Varices of œsophagus, 138
 Variola vera, 312
 Varioloid, 316
 Vasoconstrictor nerves, 215
 Vasodilator nerves, 215
 Vasomotor disorders, 215
 nerves, 215
 Vater, ampulla of, 135
 Vegetations, 49
 Veins, 11
 Ventilation of lungs, 68
 Ventricle, 183
 Vermiform appendix, 108
 Vesicle, 312

- Villi, 111
- Virulent parasites, 230
- Vomit, black, 321
- Vomiting, fecal, 121
 - nervous, 105
- Waxy casts, 153
- White blood-cells, 20
- White swelling, 280
- Whooping-cough, 260
- Widal test, 245
- Winter cough, 80, 260
- Woolsorters' disease, 296
- Work of heart, 37
- Wrist drop, 205
- Wry-neck, 194
- Yeast, 232
- Yellow fever, 320
- Zona, 205
- Zoster, herpes, 205



S.A.55.
Essentials of medicine; a text-1908
Countway Library AQK3554



3 2044 045 043 247

COUNTWAY LIBRARY



HC 2V19 T

6.A.55.

Essentials of medicine; a text-1908

Countway Library

AGK3554



3 2044 045 043 247